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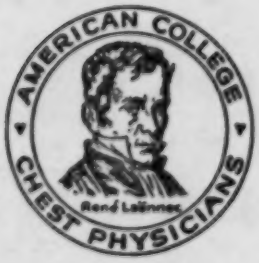
VOLUME XXVII NUMBER 3

DISEASES

of the

CHEST

OFFICIAL PUBLICATION



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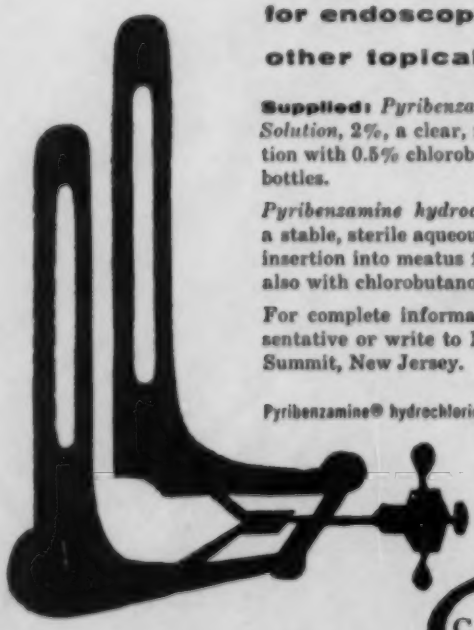

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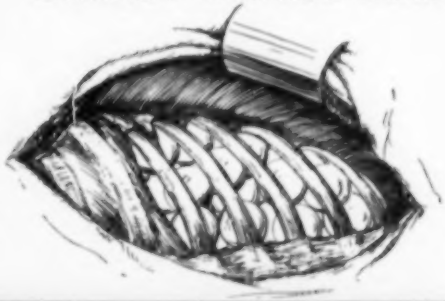

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CONTENTS

SYMPOSIUM ON THE GRAPHIC DIAGNOSIS OF CORONARY DISEASE

TYPICAL AND ATYPICAL ELECTROCARDIOGRAMS IN MYOCARDIAL INFARCTION CAUSED BY ACUTE CORONARY OCCLUSION AND CORONARY INSUFFICIENCY 243
Harry L. Jaffe, M.D., New York, New York

CARDIAC DIAGNOSTIC METHODS IN CORONARY ARTERY DISEASE: ELECTROCARDIOGRAM, "2-STEP" EXERCISE TEST AND BALLISTOCARDIOGRAM 255
Leon Pordy, M.D., New York, New York

THE VECTORCARDIOGRAPHIC DIAGNOSIS OF MYOCARDIAL INFARCTION 263
Louis Wolff, M.D., Boston, Massachusetts

THE VENTRICULAR PULSATIONS IN MYOCARDIAL INFARCTION; A FLUOROSCOPIC AND KYMOGRAPHIC STUDY 282
Simon Dack, M.D., New York, New York

REMARKS ON THE GRAPHIC DIAGNOSIS OF CORONARY DISEASE 298
Arthur M. Master, M.D., New York New York

PULMONARY FUNCTION IN DISEASES OF THE CHEST 303
Hurley L. Motley, Ph.D., M.D., Los Angeles, California

THE TIME FACTOR IN ANESTHESIA 311
Ruth Weyl, M.D., Chicago, Illinois

PALLIATIVE PROCEDURES FOR THE TREATMENT OF CARCINOMA OF THE ESOPHAGUS 316
Foster Hampton, Jr., M.D., Chattanooga, Tennesse

FLUOROSCOPY OF THE CHEST 322
Benjamin Felson, M.D., Cincinnati, Ohio

COMBINED ACTION OF PAS AND CHLOROMYCETIN ON TUBERCLE BACILLI 330
K. C. Gupta and R. Viswanathan, M.D., Delhi, India

TEACHING PHYSICAL DIAGNOSIS OF THE CHEST 333
Theodore H. Noehren, M.D. and Joseph B. Kopp, M.D., Buffalo, New York

THE PRESIDENT'S PAGE 335

21st ANNUAL MEETING PROGRAM 336

CHAPTER NEWS 355

CALENDAR OF EVENTS xxiv

MEDICAL SERVICE BUREAU xxviii

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*L. J. Cass et al., *New England J. Med.*, 249:132, 1953; *Am. J. M. Sc.*, 227:291, 1954.

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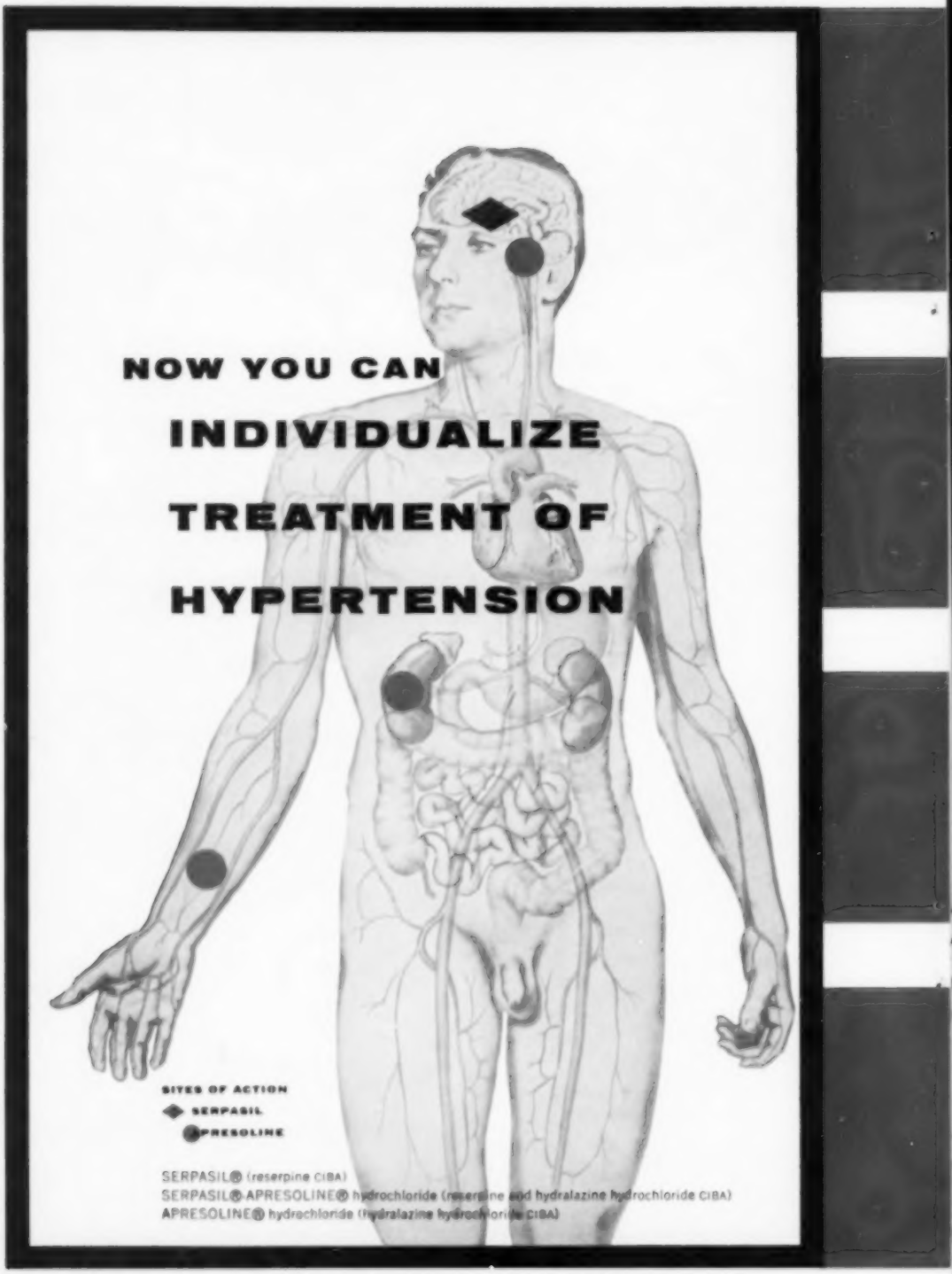
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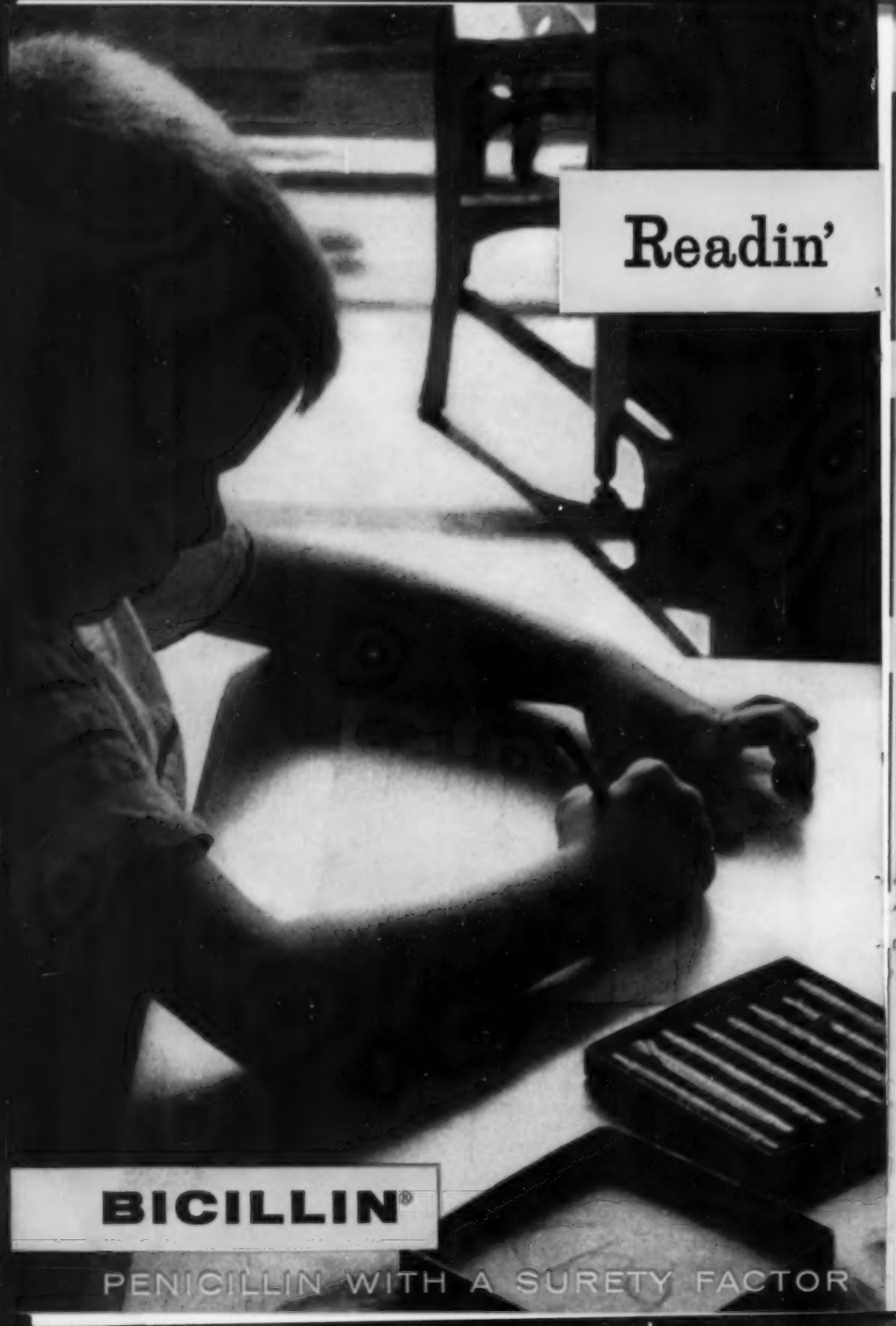
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1. Tidwell, R. A.: Northwest Med. *53*:470 (May) 1954
2. Diehl, A. M., and others: J.A.M.A. *155*:1466 (Aug. 21) 1954
3. Stollerman, G. H.: Am. Pract. *5*:589 (Aug.) 1954

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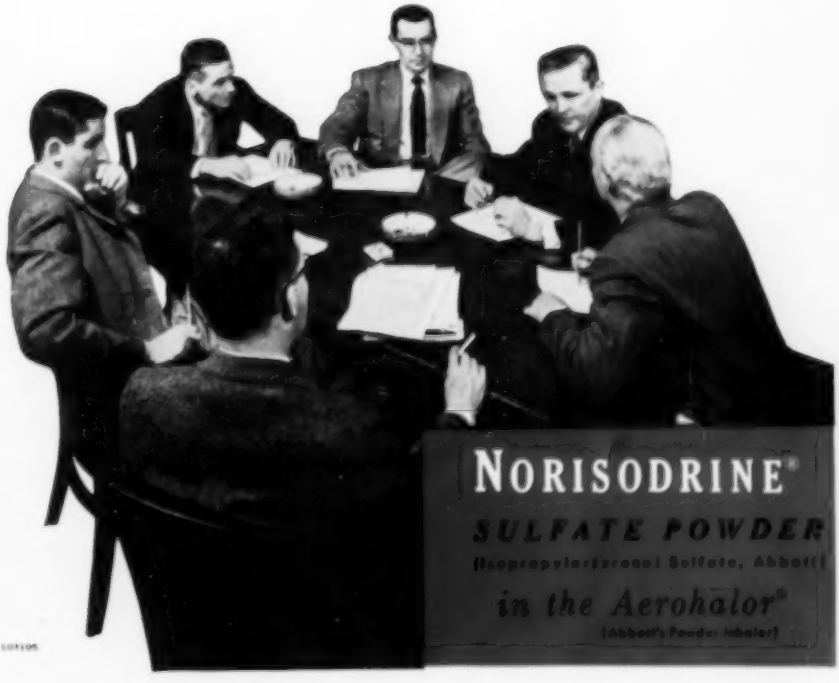

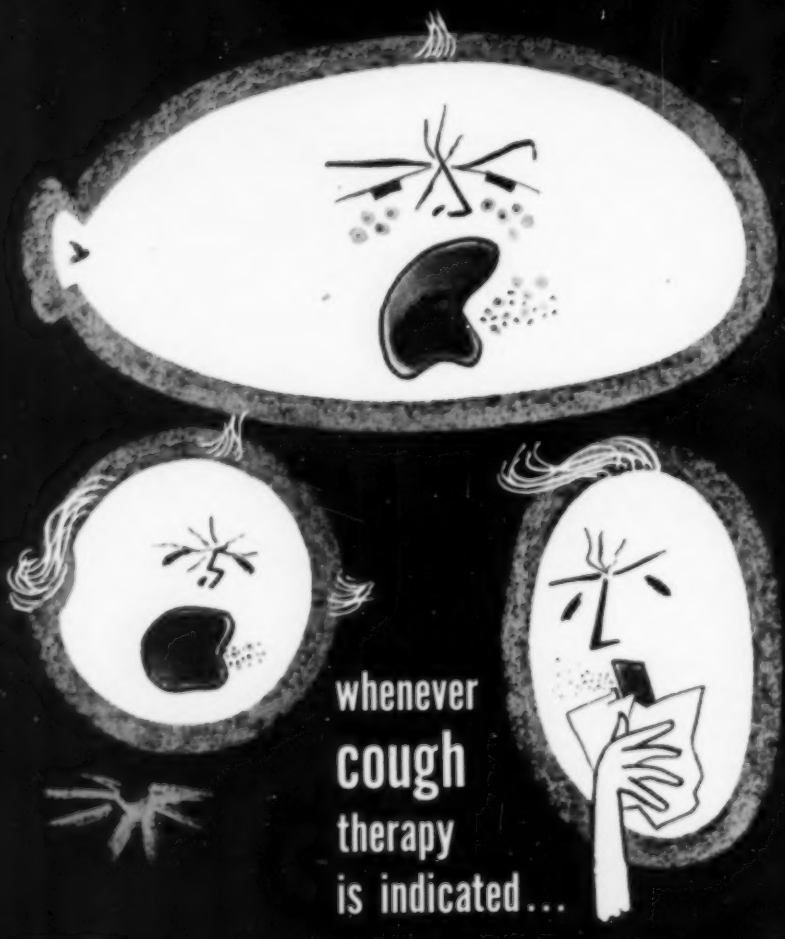

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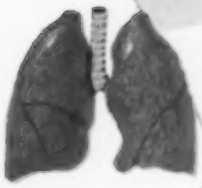

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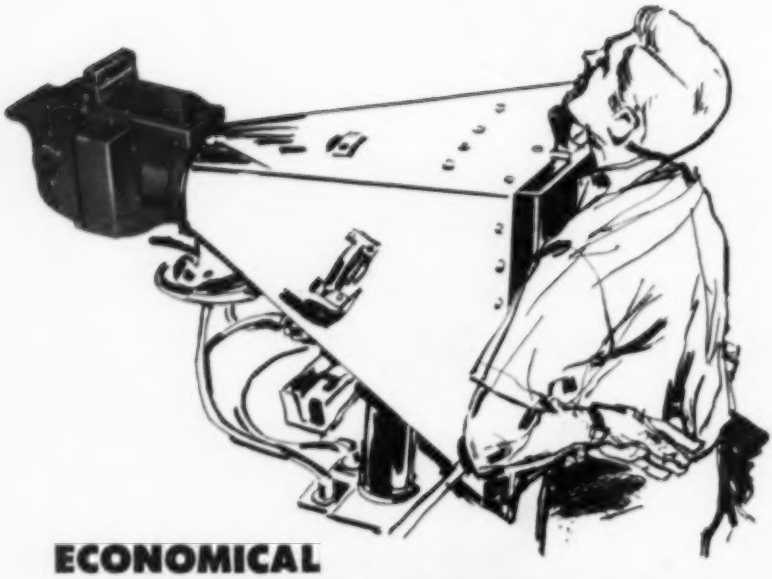

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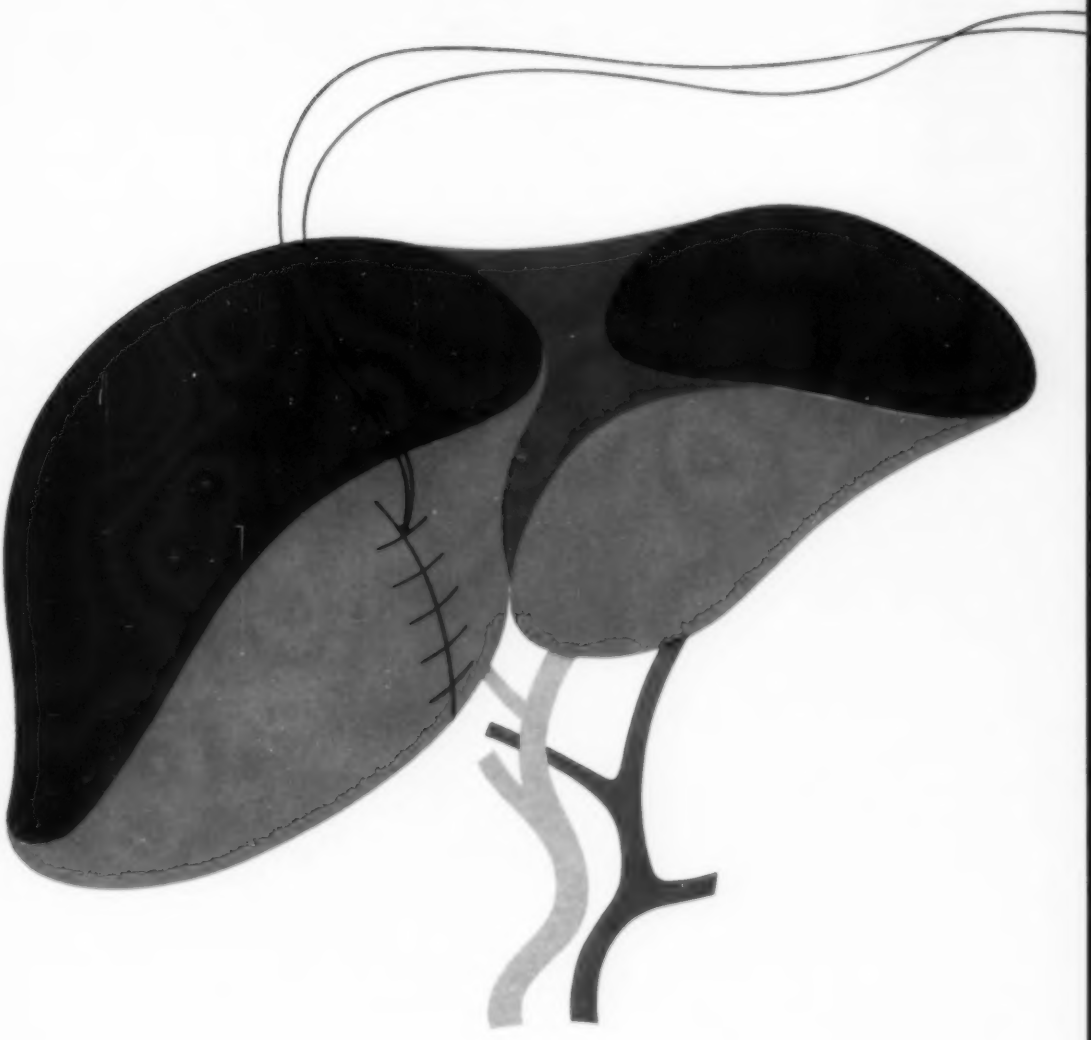

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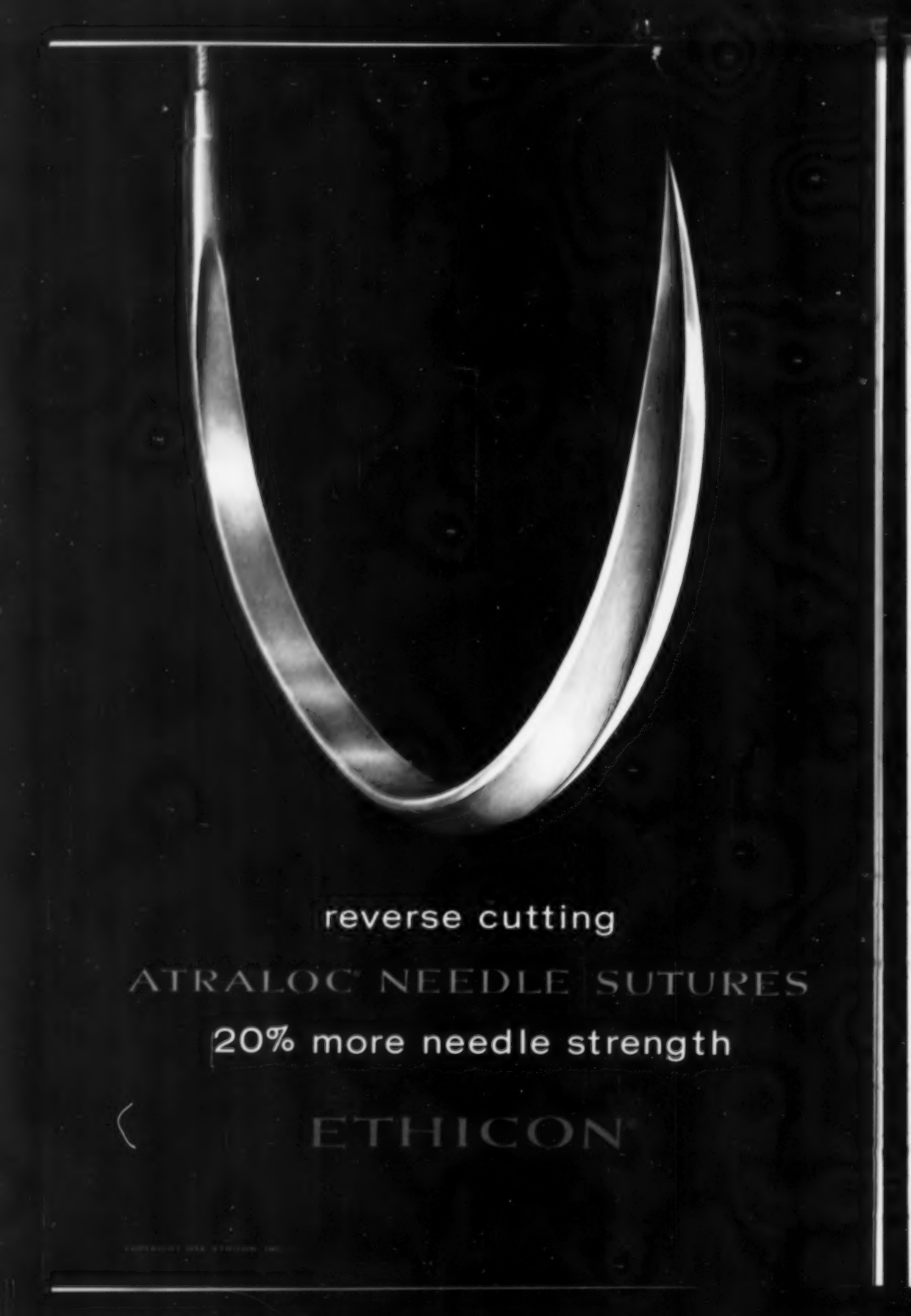
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**Heck, W.E.; Lynch, W.J., and Graves, H.L.: Acta oto-laryng. 41:416, 1953.*

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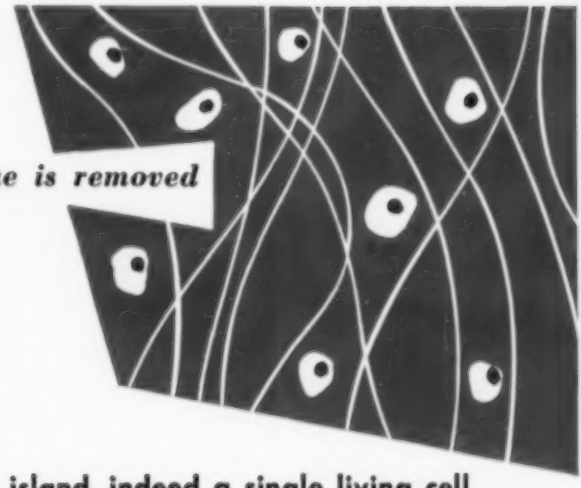

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Singer, J. J.: Differential Diagnosis of Chest Diseases, Philadelphia, Lea & Febiger, 1949, pp. 1953-4.

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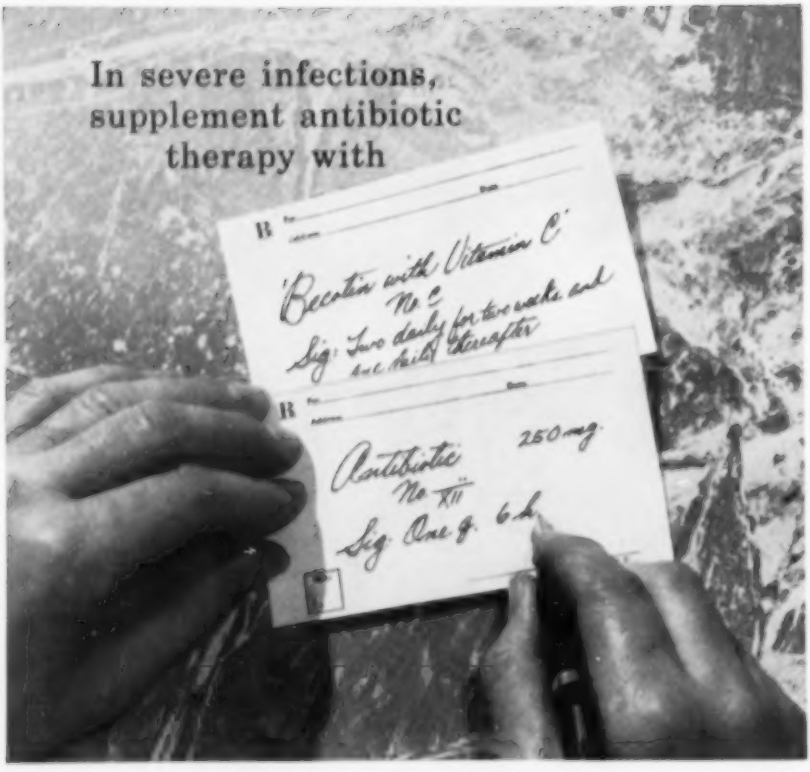
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DISEASES of the CHEST

VOLUME XXVII MARCH, 1955 NUMBER 3

Typical and Atypical Electrocardiograms in Myocardial Infarction Caused by Acute Coronary Occlusion and Coronary Insufficiency*

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New York, New York

Acute coronary episodes vary a great deal in severity and pathologically.[1-5] On the one hand there are attacks of intense, prolonged pain beginning at rest, accompanied by a drop in blood pressure, gallop rhythm, shock or congestive failure which almost always indicate a coronary occlusion with massive infarction. At the other end of the scale are the mild episodes in which the pain is of short duration and in which there are no changes in cardiac function and blood pressure. These attacks are usually caused by coronary insufficiency with either ischemia or necrosis of the subendocardial area. However, not infrequently coronary occlusion with major infarction also produces mild symptoms or the acute attack is preceded by one or more attacks of pain indistinguishable from coronary insufficiency.[3, 6-9] The differential diagnosis depends largely on the electrocardiographic findings. In over 90 per cent of cases, because they result in different types of infarction, coronary occlusion and insufficiency produce characteristic electrocardiographic patterns. As might be expected, there are numerous atypical changes, particularly as the coronary occlusion is forming and finally causes complete obstruction.

Typical Changes

Acute Coronary Insufficiency: Mild degrees of coronary insufficiency produce subendocardial ischemia; more severe forms cause focal necrosis or infarction of the subendocardial layer.[1-4] Since the ischemia or necrosis is disseminated, the electrocardiogram shows RS-T depressions and/or T-wave inversions in several or all leads. In subendocardial ischemia, the changes in the electrocardiogram revert to normal within hours or several days. In subendocardial necrosis, they persist for several weeks, months, or at times, longer.

The following two cases illustrate this difference:

Case 1: A. S., a 48-year-old business executive, suddenly experienced precordial pain and an electrocardiogram one hour later showed slight ST depression in V_5 (Fig. 1). Several minutes later the pain recurred and the depression in this lead increased. Two days later the electrocardiogram showed changes in the T-wave in leads 1 and V_{5-6}. The physical examination, blood pressure, temperature and sedimentation rate were normal and remained so. On the eighth day the electrocardiogram had returned

*From the Cardiographic Laboratory, The Mount Sinai Hospital, New York.

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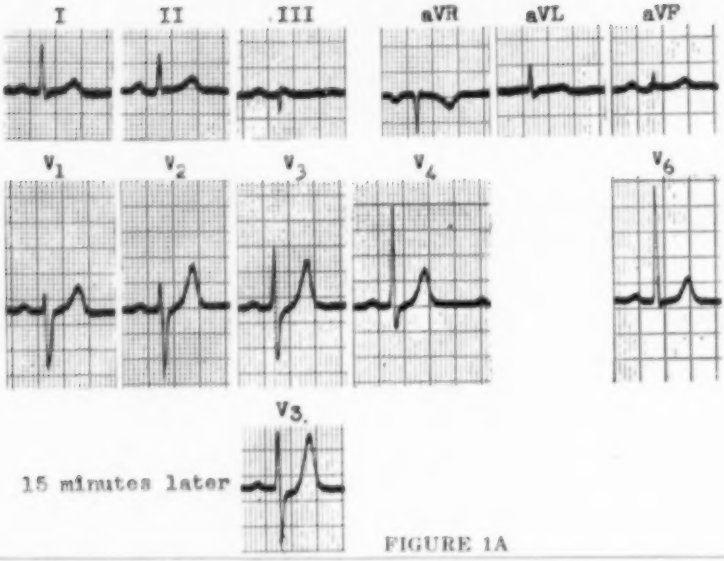

FIGURE 1A

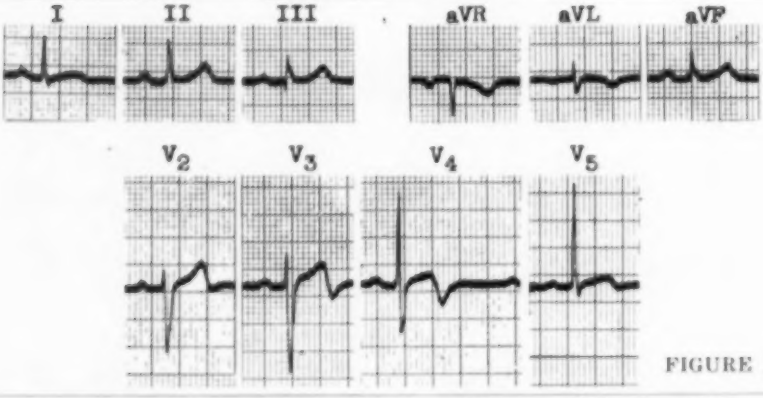

FIGURE 1B

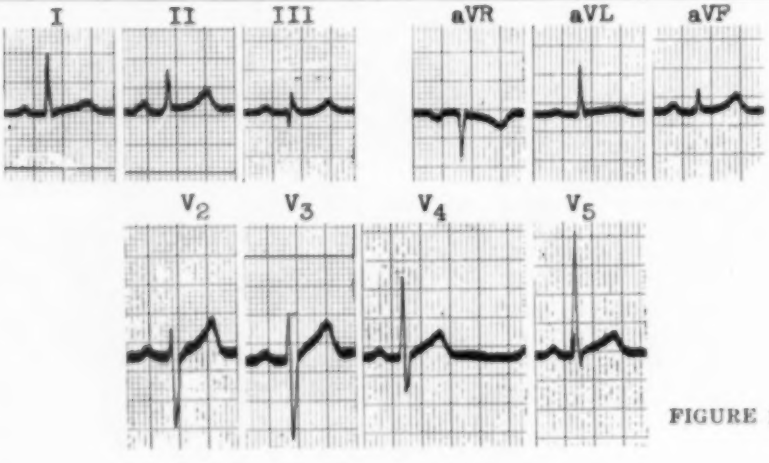

FIGURE 1C

←——— *(At Left)*

FIGURE 1A: A. S., m, 48. Acute coronary insufficiency. One hour after attack minimal ST depression present in V_5. This depression increased during recurrence of pain.—*FIGURE 1B:* A. S., m, 48. Acute coronary insufficiency. Record taken 2 days after acute attack. Temperature and sedimentation rate normal.—*FIGURE 1C:* A. S., m, 48. Eighth day; record normal.

to normal. He was permitted out of bed in one week and has been well during the two years which have elapsed since the attack.

The failure of the heart sounds, blood pressure, temperature or sedimentation rate to change stamps this as a case of coronary insufficiency with subendocardial ischemia, without significant necrosis.

Case 2: R. E., an athletics teacher, developed precordial pain for the first time at 37. It lasted two hours, after which he felt "swell." He visited the clinic on the third day and an electrocardiogram showed T-wave inversion in leads II and III (Fig. 2). His temperature was 101° and his W. B. C. was 14,000, indicating subendocardial necrosis. Two days later the electrocardiogram was within normal limits but on the ninth day of the attack it showed inversion of the T-wave in all leads which progressed during the next 11 days. His course was uneventful.

Although clinically mild, this was a definite case of subendocardial infarction with persistent T-wave inversion.

In some of these cases the T-wave changes may not appear for one or two weeks after the clinical attack, even though the sedimentation rate had become normal after several days.

Since infarction secondary to coronary insufficiency is usually not extensive, the prognosis is excellent as a rule. In other words, when the electrocardiogram shows only T-wave inversions, the patient almost always recovers.[10]

Occasionally coronary insufficiency produces temporary RS-T elevation instead of depression, thus at first simulating acute coronary occlusion with major infarction.[11] This is true, particularly, of the precordial leads in

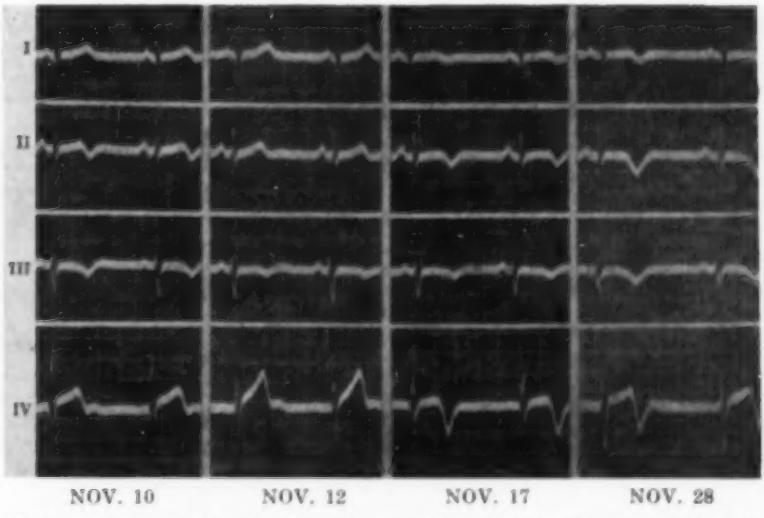

FIGURE 2: E. R., male, 37. November 10-sinus bradycardia, rate 60 beats per minute. Left axis deviation. T-II diphasic, T-III inverted. November 12—T-II now upright, T-III only slightly inverted. November 17—T-I diphasic, T-II, T-III, and T-IV inverted. November 28—T-waves definitely inverted in all leads, indicating myocardial infarction.—*From Jaffe, Master and Kalter, New York State Journal of Medicine, 1945.*

patients with previous anterior infarction; for example, if such a patient develops tachycardia, spontaneously or after exertion, the electrocardiogram may present distinct RS-T elevation, in addition to the chronic Q-wave. As soon as the tachycardia remits, the RS-T elevation disappears.

Coronary Occlusion: Characteristically, coronary occlusion produces a

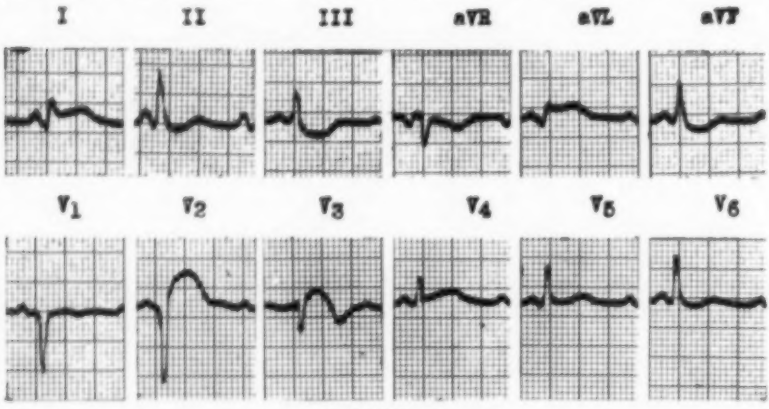

FIGURE 3A

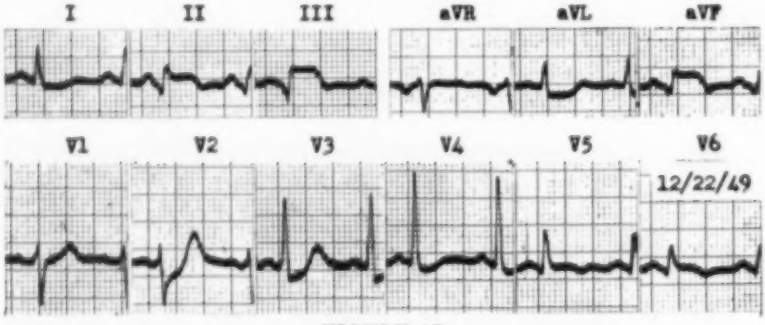

FIGURE 3B

Figure 3A: A. M., m, 53. Acute coronary occlusion. Q-waves in I, aVL, V_{1-2} with corresponding RS-T elevations. *Figure 3B:* J. S., m. 58. Acute coronary occlusion. Posterior.

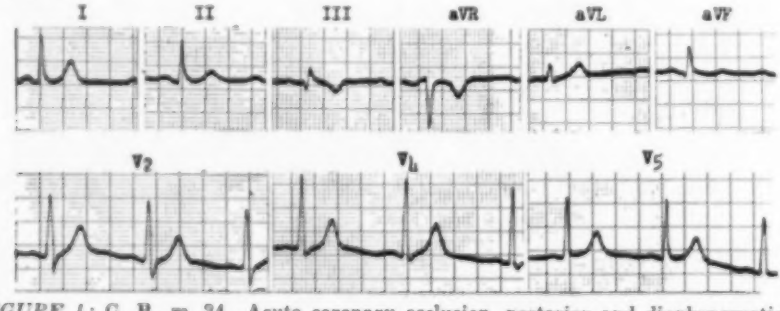

FIGURE 4: G. B., m, 34. Acute coronary occlusion, posterior and diaphragmatic infarction. A Q-wave and T-wave inversion developed in lead III but not in aVF. Note the tall R in V_2.

massive, through and through infarct and the electrocardiogram presents RS-T elevations and Q-waves (Fig. 3). The time of appearance of these changes is variable. In occasional cases the electrocardiogram remains

FIGURE 5A

FIGURE 5B

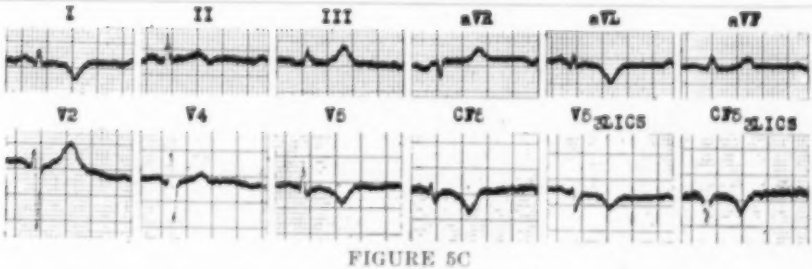

FIGURE 5C

Figure 5A: L. W., m. 64. Coronary occlusion with high lateral wall infarction, shown by CF and aVL leads but not by V leads. Record taken 10 hours after onset.—*Figure 5B:* L. W., m, 64. Coronary occlusion with high lateral infarction, Q-wave present in aVL and CF but not in V leads. Record taken 30 hours after onset.—*Figure 5C:* L. W., m, 64. Coronary occlusion with high lateral wall infarction. Two weeks after onset.

normal or unchanged for six to 10 hours, in spite of severe, persistent pain. Rarely both RS-T elevation and Q-waves appear within an hour or two after the acute attack. In many cases in which an electrocardiogram is taken within several hours, only marked RS-T elevation is present and the Q-waves do not appear until 12 to 20 hours later.

In which leads these changes appear depends upon the site and extent of the infarct. If the anterior surface of the left ventricle is infarcted, the Q-waves and RS-T elevations appear in leads 1, aVL and the precordial leads (Fig. 3A). In diaphragmatic (usually termed posterior) infarction, leads 3 and aVF shows these changes (Fig. 3B), although in some cases the Q-wave appears in lead 3 and not in lead aVF (Fig. 4). An anterior infarct may be localized to the septal area, in which case the Q and RS-T elevations appear only in V_1 to $V_{3, 4}$, or to the lateral surface, in which case these changes are usually present only in leads 1, aVL and $V_{5, 6}$ or at times only in leads 1 and aVL. In some cases it is necessary to take special precordial leads other than V_1 to 6 in order to obtain the characteristic changes. This is illustrated in Fig. 5. The patient, a merchant of 58, developed moderately severe substernal pain and a record taken 10 hours later showed only slight RS-T and T-wave changes in the routine unipolar chest leads and in those taken in the third left interspace. However, CF leads in the routine positions showed RS-T elevation and in the third interspace a large Q-wave as well. In this case, the optimum chest lead was CF and the best position the third interspace. Of course, characteristic changes were present in aVL from the start.

Frequently, the infarct extends from one surface of the left ventricle on to another and the typical Q-waves and RS-T elevations appear in various combinations of leads, e. g., in an infarct involving the diaphragmatic and anteroseptal surfaces, they occur in leads 3, aVF and V_1 to $_4$. Involvement of several areas is much more common than has usually been stated and results in considerable variation of the electrocardiographic patterns in coronary occlusion.

In coronary occlusion the electrocardiogram frequently shows reciprocal RS-T and T changes in various leads, e. g., between leads 1 and 3, aVL and aVF and, occasionally, between $V_{1, 2}$ and $V_{5, 6}$ (Fig. 3). This reciprocal relationship is obscured when pericarditis is present or when both the anterior and diaphragmatic surfaces are infarcted.

During the acute stage of infarction, the RS-T and T-waves show progressive, serial changes: the RS-T elevation gradually diminishes and disappears after two to four weeks (unless a ventricular aneurysm forms) and the T-wave becomes increasingly inverted for three to six weeks. The Q-wave usually shows little alteration during the acute stage although, occasionally, it disappears within a month or two.

In our experience, prognosis during the acute stage of coronary occlusion is not determined by the magnitude of the electrocardiographic changes. Many patients with large Q-waves and deeply inverted T-waves in all chest positions, and even some with persistent RS-T elevation for a number of weeks, run a benign course and become ambulatory in the

usual three or four weeks. Similarly, we have not found the sedimentation rate an accurate index of the severity of the attack.

Atypical Electrocardiograms

As might be expected, there are exceptions to these rules. In 5 to 10 per cent of cases coronary insufficiency produces a large infarct with RS-T elevation and Q-waves, whereas coronary occlusion produces an intramural (rather than a through and through) infarct and the electrocardiogram shows only T-wave abnormalities. An atypical electrocardio-

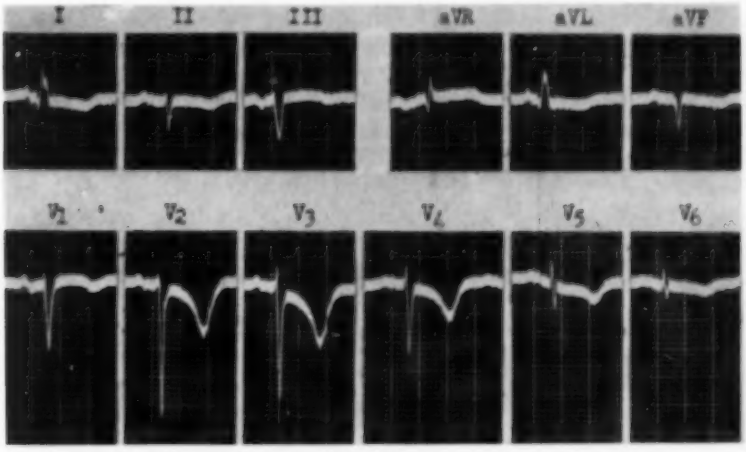

FIGURE 6A

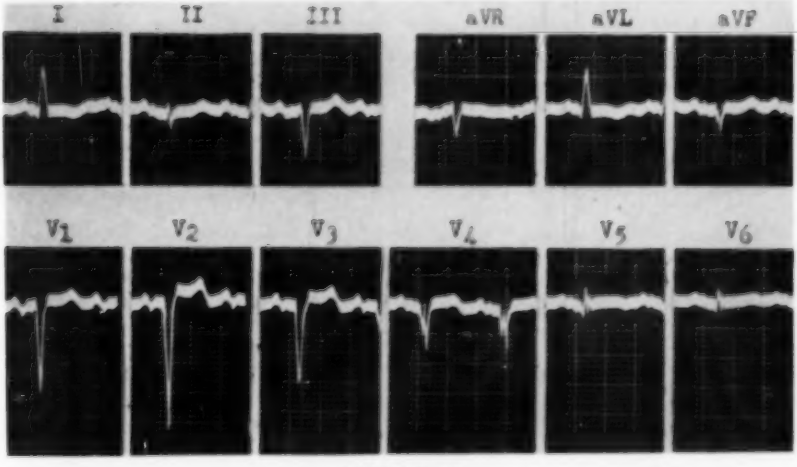

FIGURE 6B

Figure 6A: R. K., *f*, 60. Premonitory phase of coronary occlusion. Precordial pain for 2½ days. Temperature, 99.6; WBC 22,000; Polys 82 per cent. Record shows RS-T depressions and T-wave inversions.—*Figure 6B:* R. K., *f*, 60. Acute coronary occlusion with antero-septal infarction after 8 days of precordial pain. Temperature, 100. Q-waves and RS-T elevations now present in chest leads.

gram is also found in infarction of the true posterior surface (as opposed to diaphragmatic infarction which is ordinarily termed "posterior"). In such infarction a tall R-wave and tall T-waves are often present in $V_{1,2}$ and in later stages the tall R-wave may be the only indication of previous infarction (Fig. 4).[12, 13] To be sure, a tall R-wave in V_1 is often seen as part of the typical pattern of a massive antero-lateral infarct and it must also be remembered that such an R-wave is present occasionally in normal persons and frequently in right ventricular enlargement.

The Premonitory Phase of Coronary Occlusion: Another difficulty in immediately diagnosing the exact nature of acute coronary episodes results from the fact that coronary occlusion with major infarction is often preceded by an attack of pain or recurrent anginal pain at rest or on effort over a period of days or weeks.[3, 6, 9] During this period the electrocardiogram may be normal or remain unchanged, but usually it shows the changes of coronary insufficiency, i. e., RS-T depression and/or T-wave inversion (Fig. 6). In other words, many cases of coronary occlusion begin as acute coronary insufficiency, with subendocardial ischemia or necrosis, and at the onset are indistinguishable from the two cases outlined above. However, after a number of days or weeks, the patient develops a more acute attack and Q-waves and RS-T elevations appear on the electrocardiogram. Occasionally, however, these changes appear without an acute attack or even when the pain has subsided. It should be emphasized that the majority of cases of acute coronary insufficiency which fit the description of impending coronary occlusion gradually subside without going on to a major infarction.

In a few cases the coronary insufficiency changes, i. e., RS-T depression and/or T-wave inversion, persist for a day after the coronary occlusion attack has occurred, before being succeeded by RS-T elevation and Q-waves. Occasionally such changes are present temporarily after the acute occlusion even when there has been no premonitory stage (Fig. 7).

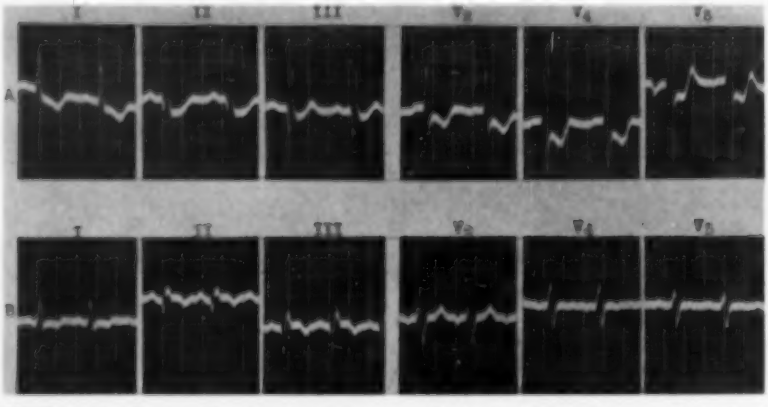

FIGURE 7: L. F., m, 63. Acute coronary occlusion with diaphragmatic infarction. Angina pectoris for eight years with probable previous infarction. *A*, One hour after acute attack of pain. *B*, Twelve hours later.

Another type of electrocardiographic change occasionally encountered at the very onset of coronary occlusion, i. e., very tall T-waves,[14] is shown in Fig. 9. This record was taken within one hour after the onset of severe pain in the mid-back. At this time the RS-T segments were still depressed. In some cases this type of T-wave persists for some hours until the RS-T has become elevated but usually the T has diminished in size by this time or by the time Q-waves have appeared.

Temporary Improvement of Electrocardiogram: A number of years ago we pointed out that in myocardial infarction RS-T and T-wave changes may temporarily improve between the third and tenth day after the attack, and the electrocardiogram may appear normal.[10, 15] At that time we considered this an unusual occurrence, but since then we have observed it more frequently. It is our impression that it occurs in approximately 5 per cent of cases. This finding is of some practical importance

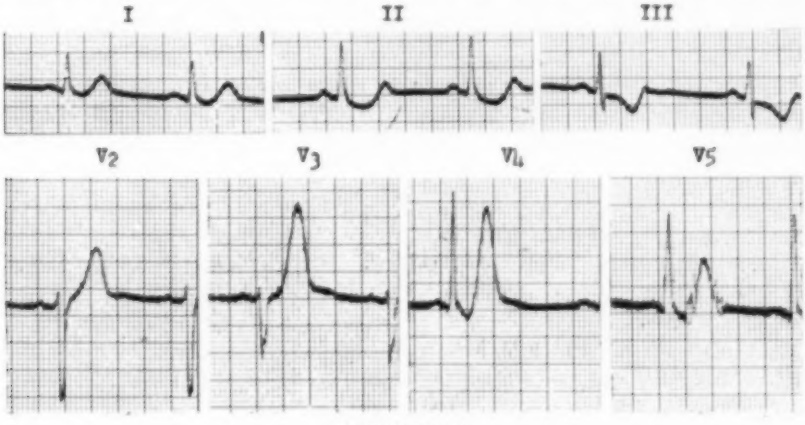

FIGURE 8A

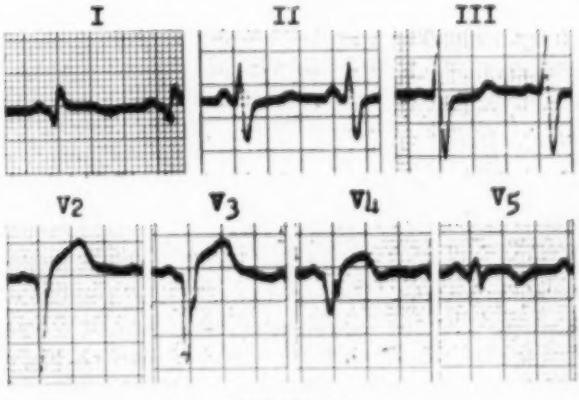

FIGURE 8B

Figure 8A: H. S., m, 51. Coronary occlusion. One hour after the onset of severe posterior chest pain. No previous symptoms.—*Figure 8B:* H. S., m, 51. Coronary occlusion with antero-septal infarction, fifth day.

for two reasons. The first electrocardiogram may not be taken until after the third day of the attack; if it is temporarily normal, the correct diagnosis may be missed. Secondly, the temporary improvement in the electrocardiogram may lead the physician to permit the patient to become active prematurely. Therefore, when the electrocardiogram improves rapidly following a coronary episode, this should be confirmed in several records.

Temporary return of the electrocardiogram to normal is illustrated in Fig. 2 and the case history has been outlined. The mechanism of this electrocardiographic finding is not clear. Such a change, i. e., an inverted T-wave becoming upright, may signify extension of the myocardial ischemia or necrosis, but in our cases there has been nothing in the clinical course to indicate this. From a similar point of view, it has been suggested that the electrocardiographic "improvement" occurs at the time when the blood pressure has fallen considerably, resulting in coronary ischemia. This, too, did not seem to be a significant factor in our cases. For example, in the case cited, the blood pressure was 130/96 at the time the electrocardiogram returned to normal, and fell moderately only several days later. Pericarditis has also been suggested as an explanation for the temporary improvement[16] but this would not account for transitory disappearance of RS-T elevation which we have observed in some cases.

Comment

We have indicated some of the changes in the E.C.G. encountered in acute coronary episodes. The most interesting cases are the mild or moderately severe attacks with only RS-T depressions and/or T-wave inversions in the E.C.G. This type of attack may follow one of these courses: 1) The E.C.G. returns to normal (or to its previous pattern) in a few days and there is no laboratory evidence of infarction or necrosis, i. e., the temperature and sedimentation rate remain normal. In these cases the pain and temporary E.C.G. changes are the result of subendocardial ischemia. 2) The E.C.G. changes persist for several weeks or months and there is some fever and elevation of the sedimentation rate during this period. In these cases subendocardial necrosis is present and the outlook is excellent. 3) The patient continues to experience recurrent pain during which the RS-T depression and T-wave changes may vary considerably and the sedimentation rate may be elevated. After a period of hours, days or weeks a severe attack develops, indicating coronary occlusion. The RS-T depression present during the premonitory phase may persist during the day after the occlusion, or very tall T-waves may appear temporarily. This is usually followed by marked RS-T elevation and, later, by Q-waves, the typical findings in massive infarction.

The last group of cases emphasize the fact that it often takes days or weeks for a thrombus to cause complete obstruction of a coronary artery. As the thrombosis becomes complete, we have seen how the electrocardiogram may show various quickly changing patterns. There is some difference of opinion as to how the process of coronary thrombosis is initiated. Some authors believe that usually it does not start directly on a sclerotic plaque but is secondary to a subintimal hemorrhage which has resulted in

injury to the intima.[9] Such a hemorrhage may also result in a hematoma which obstructs the lumen.

These subintimal hemorrhages are a common occurrence in sclerotic arteries and may cause diminution in the coronary flow, i. e., coronary insufficiency, without producing changes in the intima or thrombosis. This is probably the underlying mechanism in many of the cases in Groups 1 and 2 with ST depression and/or T-wave changes. However, many cases in these groups, particularly in Group 1 in which the electrocardiographic changes are transitory, are probably instances of coronary insufficiency precipitated by physical and emotional strain. And in some of the cases in Group 2, with persistent T-wave inversion, a coronary thrombosis with localized infarction is probably present. Because the infarct is not extensive, there are no Q-waves or RS-T elevations.

SUMMARY

When an acute coronary episode occurs, if the initial electrocardiogram does not indicate a coronary occlusion, it may be difficult at first to determine the exact nature of the attack. The electrocardiogram may remain normal for a variable period or may indicate coronary insufficiency and myocardial ischemia. In the cases going on to coronary thrombosis and massive infarction, the electrocardiogram may show various rapidly changing patterns before the appearance of RS-T elevation and Q-waves. In coronary occlusion the electrocardiogram often shows involvement of more than one surface and variations of the typical patterns are frequent. The changes may be present in only a few of the 12 routine leads or only in special chest positions.

In an acute coronary episode it is essential to keep the patient at rest until the extent of myocardial involvement has been determined.

Not infrequently the electrocardiogram shows temporary improvement or actual return of RS-T and T to normal between the third and 10th days following myocardial infarction.

RESUMEN

Cuando ocurre un episodio coronario agudo, si el electrocardiograma no indica una oclusión coronaria, ser difícil al principio elsentar un diagnóstico. El electrocardiograma puede permanecer normal por un período variable o puede indicar insuficiencia coronaria e isquemia del miocardio. En los casos que marchan hacia la trombosis coronaria y el infarto masivo, el electro puede mostrar varios aspectos rápidamente cambiantes antes de la elevació RS-T aparezca así comolas ondas Q.

En la oclusión coronaria el electro a menudo muestra compromiso de más de una superficie y las variaciones del aspecto típico son frecuentes.

Los cambios pueden presentarse en sólo pocos de los doce puntos derutina o sólo en posiciones especiales del tórax.

En un episodio agudo coronario es esencial conservar el enfermo enreposo hasta que la extensión del daño miocárdico se haya determinado.

No espoco frecuente que el electrocardiograma muestre una mejoríatemporal o verdadero regreso de RS-T y T a la normal el tercero y el décimo día después del infarto del miocardio.

RESUME

Lors d'un épisode coronarien aigu, si l'électrocardiogramme n'indique pas dès le début l'existence d'une occlusion coronarienne, il peut être difficile de déterminer la nature exacte d'une telle attaque. L'électrocardiogramme peut rester normal pendant un période de temps variable ou peut indiquer une insuffisance coronarienne avec ischémie myocardique. Dans les cas où l'évolution se fait vers la thrombose coronarienne et l'infarctus massif, l'électrocardiogramme peut montrer des aspects variés et rapidement changeants, avant que n'apparaisse l'élévation de R.S.T. et de l'onde Q. Dans l'occlusion coronarienne, l'électrocardiogramme montre souvent des altérations de plusieurs parois et les variations du dessin classique sont fréquentes. Les altérations peuvent n'exister que sur un petit nombre des douze dérivations classiques ou bien uniquement dans le cas de position particulière du thorax.

Lors d'un épisode aigu, il est essentiel de laisser le malade au repos jusqu'à ce que soit déterminée l'étendue des lésions myocardiques.

Il n'est pas exceptionnel de constater à l'électrocardiogramme une amélioration ou bien un retour à la normale des ondes R.S.T. et Q entre le troisième et le dixième jour suivant la constitution de l'infarctus myocardique.

REFERENCES

1 Master, A. M., Gubner, R., Dack, S. and Jaffe, H. L.: "Differentiation of Acute Coronary Insufficiency with Myocardial Infarction From Coronary Occlusion," *Arch. Int. Med.*, 67:647, 1941.

2 Master, A. M., Jaffe, H. L., Dack, S. and Grishman, A.: "Coronary Occlusion, Coronary Insufficiency, and Angina Pectoris," *Am. Heart J.*, 27:803, 1944.

3 Jaffe, H. L., Halprin, H. and Nelson, L. M.: "Evaluation of Anginal Pain in Various Stages of Coronary Artery Disease, Particularly the Premonitory Phase of Coronary Occlusion and Infarction without Occlusion," *N. Y. State J. Med.*, 47: 1383, 1947.

4 Master, A. M. and Jaffe, H. L.: "Coronary Occlusion and Coronary Insufficiency," *Postgrad. Med.*, 9:279, 1951.

5 Jaffe, H. L.: "Unusual Electrocardiograms Before and During Acute Coronary Occlusion and Myocardial Infarction," *Program, 26th Scientific Session, American Heart Assn.*, (Apr. 9-12) 1953, p. 6.

6 Master, A. M., Dack, S. and Jaffe, H. L.: "Premonitory Symptoms of Acute Coronary Occlusion: A Study of 260 Cases," *Ann. Int. Med.* 14:1155, 1941.

7 Sampson, J. J. and Eliaser, M. Jr.: "Diagnosis of Impending Acute Coronary Artery Occlusion," *Am. Heart J.*, 13:675, 1937.

8 Jaffe, H. L.: "Electrocardiographic Changes of Coronary Insufficiency During the Formative Stage of Coronary Occlusion," *J. Mt. Sinai Hosp.*, 16:404, 1950.

9 Horn, H. and Finkelstein, L.: "Arteriosclerosis of the Coronary Arteries and the Mechanism of Their Occlusion," *Am. Heart J.*, 19:655, 1940.

10 Jaffe, H. L., Master, A. M. and Kalter, H.: "An Unusual Electrocardiographic Finding in Myocardial Infarction," *N. Y. State J. Med.*, 45:1565, 1945.

11 Kropp, I. G., Jaffe, H. L. and Master, A. M.: "Significance of RS-T Elevation in Acute Coronary Insufficiency," *Bull. N. Y. Acad. Med.*, 24:465, 1949.

12 Grishman, A. and Scherlis, L.: "Spatial Vectorcardiography," *Saunders Co.*, Phila. 1952, p. 162.

13 Grishman, A., Jaffe, H. L. and Scherlis, L.: "The Electrocardiogram in True Posterior Infarction," To be published.

14 Dressler, W. and Roesler, H.: "High T-waves in the Earliest Stages of Myocardial Infarction," *Am. Heart J.*, 34:627, 1947.

15 Jaffe, H. L. and Master, A. M.: "Temporary Return of the Electrocardiogram to Normal in Acute Coronary Occlusion," *Proc. Am. Federation Clin. Research* (1946) 3:70, 1947.

16 Sokolow, M.: "Personal Communication."

Cardiac Diagnostic Methods in Coronary Artery Disease: Electrocardiogram, "2-Step" Exercise Test and Ballistocardiogram

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The clinical importance and necessity of dependable cardiac diagnostic and function tests are emphasized by the outstanding fact that cardiovascular disease is the leading cause of death in the country today. However, the time honored methods of complete history, adequate physical examination, chest fluoroscopy and roentgenogram will never be replaced in value by the other available tests of heart function. The latter serve as corroborative laboratory aids for diagnosis and prognosis and in this regard, they play a vital role in the final clinical impression of the cardiac status.

Electrocardiography

Since the introduction of an accurate recording apparatus, the electrocardiograph has assumed increasing importance daily in the routine diagnosis of coronary artery disease.

Technique: Although the present electrocardiographic recording apparatuses leave much to be desired insofar as high frequency response is concerned, the customary amplifier type or string galvanometers yield records of sufficient fidelity for routine clinical use. All records should be accompanied by the standardization utilized, that is, 1 milli-Volt — 1 centimeter deflection for routine tracings. At present 12 lead records are recommended routinely: the three standard leads (I, II and III), the three augmented unipolar limb leads (aVR, aVL and aVF); and the six unipolar precordial chest leads (V_1 through V_6). Although less than the 12 mentioned are recorded routinely in many cardiac laboratories, instances of coronary disease in which abnormalities occur in only one lead are sufficiently common to preclude such abbreviations of technique. Moreover, situations such as high lateral myocardial infarction, may require even additional chest exploration for the detection of the cardiographic alteration.

The use of CR, CL or CF chest leads routinely as opposed to unipolar V leads introduces the valid objection of undue influence of the remote extremity electrode on the exploring chest piece. Current argument in favor of CF over V chest leads cannot be supported on theoretical grounds and any diagnostic advantage in an individual case may be considered merely fortuitous. On the other hand, the apparent advantage of unipolar extremity leads such as aVF in the diagnosis of posterior (diaphragmatic) myocardial infarction has been questioned by many workers.

Somatic tremor may usually be readily eliminated, but in some cases, as in Parkinsonism, may produce a regular undulation of the baseline

which may be mistaken for auricular flutter. Alternating current interference should be eliminated by proper grounding.

Interpretation: The criteria for normality and abnormality of electrocardiographic tracings as regards coronary artery disease are generally universally accepted, although individual interpretations may vary. Unfortunately, normal individuals as well as those with anxiety states may present abnormal resting electrocardiographic tracings. Ergotamine[1] and more recently, dihydroergocornine,[2] have been utilized in the differentiation of such abnormal resting electrocardiographic tracings in functional heart disturbance as compared with organic coronary artery disease, the record returning to normal during exhibition of these drugs in the former condition.

Interpretation of electrocardiographic records as regards coronary disease without adequate knowledge of the age and clinical status of the patient is fraught with danger. Refusal to render a reading of tracings without this information is actually entirely justifiable. For example, the cardiac rotation and displacement with pulmonary collapse may result in electrocardiographic "abnormalities" in the absence of cardiac involvement.

Specificity: The Q waves of myocardial infarction represent the truly specific alterations of the electrocardiogram diagnostic of complete coronary artery occlusion. Although the appearance of significant Q waves may be delayed for several days or longer after acute coronary occlusion with through and through myocardial infarction, they usually remain permanently as the indicator of previous severe myocardial damage. With subsequent episodes of acute coronary occlusion, Q waves may appear in other leads or become deeper and/or wider, if already present. Q waves are usually considered significant only when they represent 25 per cent or more of the amplitude of the R wave or when they are 0.04 second or longer in duration. It should be remembered that a deep Q or QS deflection is a normal finding in lead aVR and that myocardial infarction may be reflected in this lead by the appearance of an early R wave! Even in the absence of significant Q waves, anterior wall myocardial infarction may be diagnosed by an alteration of the normal increment of the amplitude of the R wave as one proceeds from V_1 through to lead V_6 on the chest wall. In some instances, after recovery from acute coronary occlusion, the Q waves may become smaller and some records may lose the Q wave entirely and become normal.

RS-T segment deviations and T wave alterations are *not* specific for coronary artery disease. Unfortunately, these abnormalities may be associated with many other disease states such as rheumatic heart disease, pericarditis of any etiology, cardiac hypertrophy of any cause, pulmonary embolization, etc. Moreover, electrolyte imbalance (potassium, calcium) and even drinking of ice water may produce electrocardiographic changes in the RS-T segment and/or T waves indistinguishable from those associated with organic coronary artery disease. It is important to emphasize the difference between the electrocardiographic pattern of acute coronary

insufficiency and that of acute coronary occlusion. In the former there is noted typically RS-T segment depression and T wave inversion in all leads without the appearance of Q waves except aVR where one finds RS-T segment elevation and upright T waves. These findings are related to the subendocardial ischemia of coronary insufficiency. On the other hand in acute coronary occlusion with myocardial infarction, Q waves are typical and in leads with significant Q waves the RS-T segment is usually elevated and later T wave inversions appear. In infarction of the posterior wall of the myocardium (diaphragmatic) one may note tall pointed T waves in the right chest V leads.

Bundle branch block, intermittent or permanent, left or right, complete or incomplete, may occur in the presence of coronary artery disease. These patterns may obscure the appearance of significant Q waves with the onset of acute coronary occlusion—especially anterior wall myocardial infarction in a patient with left bundle branch block. However, it should be noted that such block patterns may be functional, positional, respiratory or congenital.

In coronary artery disease, the electrocardiogram may disclose tall, wide P waves, prolongation of the P-R interval, incomplete or complete A-V block, inverted u waves, premature beats and any other arrhythmia. Again, none of the alterations are specific for coronary disease. The Wolff-Parkinson-White pattern may be found in normal individuals as well as those with organic heart disease.

Diagnostic Value: The electrocardiogram at rest has been found to be entirely *normal in from* 25 to 60 per cent of patients with organic coronary artery disease. Therefore, early in the disease one may expect a normal tracing in a large number of individuals and of necessity, one must rely on other cardiac diagnostic tests. Later on in the progress of the disease, the electrocardiogram is more apt to be abnormal and then of diagnostic value in a positive sense. It must be reemphasized that a normal or abnormal electrocardiographic tracing must be interpreted in the light of the entire clinical picture to be of full value. Moreover, when the latter is combined with serial tracings, obscure states may often be resolved correctly and thus prognostic value may be obtained in the resting electrocardiogram.

The absolute necessity for more dependable methods for the diagnosis of coronary artery disease has led to the increasing popularity of the following tests of cardiac function—the "2-step" exercise electrocardiogram[3, 4] and the ballistocardiogram.[5-8]

"2-Step" Exercise Electrocardiogram

It has been found that in 25 to 60 per cent of patients with coronary artery disease, the results of *all* routine investigations may be normal—physical examination, cardiac fluoroscopy, and chest teleroentgenogram. Following the introduction of the "2-step" exercise procedure in 1929, Master later applied the electrocardiographic changes following standard exercise as the test for coronary insufficiency.

Technique: After routine examination, the "2-step" test is performed only if the 12 lead resting electrocardiogram is normal. The patient walks over the "2-step" stairs with the electrodes left in place. Each step is nine inches (22.9 cm.) high. Routinely standard leads 1, 2, and 3 and precordial lead, V_4 or V_5 (with the tallest, widest R wave) are taken. More recently the four leads recorded are standard lead 2 and precordial leads V_3, V_4 and V_5. In the single test, the required number of ascents (complete trips over the steps in one direction) is determined from a table based upon sex, age and weight. The single test is performed in one-and-one-half minutes and the patient then returns to his original position, sitting or recumbent. The electrocardiogram (4 leads) is recorded immediately, two minutes and six minutes after completion of the exercise. In order to avoid dizziness the direction of the patient is changed after the completion of each trip; that is, he turns toward the examiner each time. In the event that the single test is normal, then a minimum of one hour later the double "2-step" is performed. The latter is twice the number of standard trips in three minutes and is advised only if the single "2-step" is normal, for reasons of safety. The importance of the double exercise is borne out by the fact that one-third of patients with abnormal double tests presented normal single tests.

Interpretation: Criteria for abnormal single and/or double "2-step" tests include: (1) Depression of the RS-T segment greater than ½ mm. below the isoelectric (P-R) level; (2) Complete or partial inversion of a T-wave (except in lead 3); or (3) transient arrhythmias, conduction defects, or large Q waves. These alterations are identical with the electrocardiographic changes during spontaneous episodes of angina pectoris. The most sensitive leads for this test are the precordial (V_4 or V_5). Tracings are repeated beyond six minutes if necessary, until the record returns to the normal control. These electrocardiographic alterations are independent of the appearance of chest pain or pressure and the latter is *not* a criterion for a positive test. The "2-step" exercise is contraindicated if heart failure, cardiac enlargement or an abnormal resting electrocardiogram is found. Food or exercise within one hour, tobacco, digitalis and nitroglycerin all influence the result of the test.

Specificity: Abnormal "2-step" exercise electrocardiograms, single or double, present definite evidence of coronary insufficiency on effort. It must be emphasized that abnormal tests are not specific for coronary artery disease since other organic or functional states may be the underlying cause (rheumatic, luetic or congenital heart disease or severe anemia, for example). On the other hand the "2-step" test may be abnormal in from 6 to 8 per cent of normal people and also in patients with neurocirculatory asthenia or anxiety states. Differentiation between such functional states and true organic heart involvement may be made usually on clinical grounds alone. However, we have found dihydroergocornine (DHO-180), a "sympatholytic" agent, of value in this regard.

Diagnostic Value: The value of the "2-step" exercise test has been corroborated by a five year follow-up study of hundreds of patients. If both

the single and double "2-step" tests are normal, a diagnosis of coronary insufficiency and hence, coronary artery disease, is practically (but not absolutely) excluded. Thus, the value of the "2-step" test in the negative sense is inestimable. On the other hand, in patients with normal resting electrocardiograms, an abnormal single or double "2-step" test presents objective evidence of coronary insufficiency. The decision of a final label of coronary artery disease rests with the examining physician after correlation of the abnormal results of the exercise with the clinical status of the patient.

Ballistocardiography

In 1949 with the introduction of the direct body recording technique by Dock and Taubman,[6] the ballistocardiograph became available generally as a practical cardiac function test. The ballistocardiogram is a record of the motion of the body induced by the contraction of the heart and the surge of blood into the arterial tree during each cardiac cycle. We have investigated the ballistocardiograph in over 2,000 patients during the past four years and have utilized a modified Dock type instrument, the Pordy dual ballistocardiograph for recording both photoelectric (displacement) and electromagnetic (velocity) tracings with a single setting. A report of the accuracy of this apparatus as checked mechanically with linear reciprocating motion by Dr. Sergei Feitelberg is in progress.

Technique: The patient lies supine on a fixed, immobile table. A crossbar placed on the shins transmits the longitudinal body motions (without a hinge) by cutting light falling on the photo-cell and/or by altering an electromagnetic field. The record is taken on the customary electrocardiographic machine by direct attachment to the lead 1 electrodes. Whenever time reference is desired, simultaneous electrocardiograms and ballistocardiograms may be recorded simply, even on the routine single channel electrocardiographs. The ballistocardiogram is recorded during quiet respiration and then deep inspiration and finally deep expiration.

Interpretation: Ballistocardiograms, like electrocardiograms, may be read by direct inspection of the record. The normal ballistocardiogram is of a W-shaped appearance, with the base-line one which bisects the waves. By convention, headward motion of the body is represented as an upward deflection on the record and *vice versa*. The component waves are labeled in alphabetical order according to Starr, commencing with the letter "H." This represents the first headward motion with each cardiac cycle and is related to the apical thrust. The I-wave represents the recoil of the body to ejection in early systole. The most prominent headward wave is the J-wave and this results from impact on the aortic arch and acceleration of blood in the aorta. The K-wave is the most prominent footward deflection and is related to deceleration in the descending aorta. L, M, N and O, and so on, are the diastolic after vibrations or actually forced thrusts.

The records are interpreted on the qualitative appearance of the component waves of the ballistocardiogram and we have not attempted their use quantitatively as a measure of cardiac output. In normal persons,

there is a respiratory variation in the amplitude of the complexes, being of greater amplitude in inspiration than in expiration. Criteria for abnormality include: (1) diminished or absent I-waves; (2) exaggeration of the normal respiratory variation so that the amplitude of the waves during expiration is 50 per cent or less than during inspiration; (3) slurring or notching of I, J, or K-waves; (4) early "M" pattern—prominent H-waves; (5) late "M" pattern—late, deeply notched J-waves; (6) prominent diastolic waves (L, M, N, O, and so on); (7) deep, wide or absent K-waves; or (8) low amplitude or totally bizarre complexes. Any one record may display one alteration alone or various combinations. Moreover, abnormal waves may be detected in only one phase of respiration. For example, in early coronary disease alterations may appear in the complexes only during the expiratory phase.

Specificity: Like the resting and "2-step" exercise electrocardiogram, there is no ballistocardiographic pattern specific for coronary artery disease. The factor of age plays an important role in interpretation. Under the age of 50 one would expect to find a normal ballistocardiogram almost routinely. Therefore, an *abnormal* tracing in a coronary disease suspect under 50 is of great significance diagnostically, since thereafter the record may be altered by physiological aging processes. On the other hand a *normal* ballistocardiogram in a patient over 50 would correlate well with a normal cardiovascular tree. It must be pointed out that in patients with coronary artery disease who undergo complete functional recovery, the ballistocardiogram and the resting as well as exercise electrocardiogram may return to normal.

Diagnostic Value: Ballistocardiography has its greatest application in the diagnosis of coronary artery disease. In patients with angina pectoris and abnormal resting electrocardiograms, 93 per cent presented abnormal ballistocardiograms as well. Moreover, in 85 per cent of patients with angina pectoris, normal resting electrocardiograms but abnormal "2-step" exercise tests, the ballistocardiograms *at rest* were abnormal. The use of standard exercise ballistocardiograms increased this figure to 90 per cent. In early cases of coronary artery disease, cardiac ejection may be altered only following exertion. Therefore, the ballistocardiogram should be recorded after standard exercise if the resting record is normal. A close correlation was found between abnormal "2-step" exercise tests and abnormal resting ballistocardiograms. Both should be employed routinely in the detection of coronary artery disease since one records electrical and the other mechanical events.

The high percentage of abnormal ballistocardiograms in hypertensive patients limits its value somewhat in the diagnosis of coronary artery disease. In patients with acute myocardial infarction, the ballistocardiogram becomes abnormal and may return to normal (in 19 per cent) usually paralleling functional recovery. Thus the value extends to prognosis in coronary disease patients. In the diagnosis of coronary disease, the ballistocardiogram offers a simple means of early detection with the advantage of lack of any danger to the patient. The use of the tobacco

ballistocardiographic test may enhance the diagnostic value of this procedure in coronary disease.

Conclusion

The clinical status and routine examination of the patient should be correlated with the results of a battery of tests—the electrocardiogram, the Master "2-step" exercise test and the ballistocardiogram—for the final, early and accurate diagnosis of coronary artery disease.

SUMMARY

1. The scope of the problem of the diagnosis of coronary artery disease is discussed.
2. The value and limitations of routine electrocardiography is outlined.
3. The Master "2-step" exercise test is described and the diagnostic value is stressed.
4. The ballistocardiogram is presented as a valuable means for early detection of coronary disease.

RESUMEN

1. Se discute el alcance del problema del diagnóstico de la afección de la arteria coronaria.

2. Se presenta el valor y las limitaciones de electrocardiografía de rutina.

3. El ejercicio "Master two step" se detalla y se subraya su valor diagnóstico.

4. Se presenta el balistocardiograma como un valioso medio parael descubrimiento temprano de la afección coronaria.

El estado clínico y el examen de rutina del enfermo deben ser correlativos con el resultado de las pruebas en batería o grupe: el electrocardiograma, el ejercicio "Master two step" y el balistrocardiograma, a fin de hacer un diagnóstico final y exacto de la enfermedad coronaria.

RESUME

1) L'auteur met en discussion la question du diagnostic de l'atteinte de l'artère coronaire.

2) Il insiste sur la valeur et sur les limites de l'électrocardiogramme habituel.

3) Il précise la valeur du test essentiel de l'exercice dit "des deux pas" et son importance au point de vue du diagnostic.

4) Il considère le ballistocardiogramme comme un moyen valable pour découvrir précocément l'atteinte coronarienne.

L'état clinique et l'examen systématique du malade doivent être conjugés á ce que donne toute une série de tests: électrocardiogramme, exercice "des deux pas," ballistocardiogramme. Ainsi le diagnostic das maladies coronariennes pourra être définitif, précoce et précis.

REFERENCES

1 Master, A. M., Pordy, L., Kolker, J. and Blumenthal, M. J.: "The 'Two-Step' Exercise Electrocardiogram in Functional Heart Disturbances and in Organic Heart Disease: The Use of Ergotamine Tartrate," *Circulation* 1:692 (April) 1950.

2 Pordy, L., Arai, H. S. and Master, A. M.: "Dihydroergocornine in the Differential Diagnosis of Functional Heart Disturbances and Organic Heart Disease," *J. Mt. Sinai Hosp.*, 17:26 (May-June) 1950.

3 Master, A. M.: "The 'Two-Step' Exercise Electrocardiogram: A Test for Coronary Insufficiency," *Ann. Int. Med.*, 32:842 (May) 1950.

4 Master, A. M., Pordy, L. and Chesky, K.: "Two-Step Exercise Electrocardiogram. Follow-Up Investigation in Patients With Chest Pain and Normal Resting Electrocardiogram," *J.A.M.A.*, 151:458 (Feb. 7) 1953.

5 Starr, I., Rawson, A. J., Schroeder, H. A. and Joseph, N. R.: "Studies on Estimation of Cardiac Output in Man and of Abnormalities on Cardiac Function, from Heart's Recoil and Blood's Impacts; Ballistocardiogram," *Am. J. Physiol.*, 127:1, 1939.

6 Dock, W. and Taubman, F.: "Some Techniques for Recording the Ballistocardiogram Directly from the Body," *Am. J. Med.*, 7:751, 1949.

7a Pordy, L., Taymor, R. C., Moser, M., Chesky, K. and Master, A. M.: "Clinical Evaluation of the Ballistocardiogram," I. Normal Subjects. *Am. Heart J.*, 42:321 (Sept.) 1951.

7b Chesky, K., Moser, M., Taymor, R. C., Master, A. M. and Pordy, L.: "Clinical Evaluation of the Ballistocardiogram." II. Heart Disease—Hypertension, Angina Pectoris and Myocardial Infarction. *Am. Heart J.*, 42:328 (Sept.) 1951.

8 Taymor, R. C., Pordy, L., Chesky, K., Moser, M. and Master, A. M.: "Ballistocardiogram in Coronary Artery Disease," *J.A.M.A.*, 148:419 (Feb. 9) 1952.

The Vectorcardiographic Diagnosis of Myocardial Infarction

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Interest in spatial vectorcardiography has grown considerably in the past 15 to 20 years, and increasing numbers of workers are investigating the method and probing its diagnostic usefulness. There is still a great deal of disagreement concerning fundamental issues, largely the result of lack of unanimity in regard to methods, and views concerning its value compared to the electrocardiogram are at considerable variance.

Preliminary experience in this laboratory led to the unqualified opinion that spatial vectorcardiography contributes to the basic understanding of the electric phenomena associated with the heart beat, and reveals diagnostic clues unknown or impossible in electrocardiography.[1] The conclusion was reached that right and left ventricular hypertrophy, singly or combined, intraventricular block, and myocardial infarction are disclosed more often and more clearly by the vectorcardiogram than by the electrocardiogram. Our continued experience in this field has strengthened this point of view. Since there are no generally accepted diagnostic criteria in vectorcardiography, this opinion is based, for the most part, on a comparison of vectorcardiographic and electrocardiographic interpretation. Consequently, the electrocardiographic interpretation, being at present the more generally acceptable, serves as the standard. A more satisfactory approach to the problem was attempted in relation to posterior myocardial infarction, and appeared to substantiate the earlier impressions.[2]

A larger study has now been completed, constituting our total experience to date. This embraces a comparison of vectorcardiographic and electrocardiographic interpretation with pathologic correlation in 50 consecutive cases which came to autopsy.[3] The data strikingly demonstrate the superiority of the vectorcardiogram. Among the 50 cases were 22 (exclusive of those with left bundle branch block) in which autopsy showed myocardial infarction, and 10 of these, selected at random, are presented below. The pertinent clinical and pathological data, and the electrocardiographic and vectorcardiographic diagnoses are listed in Table I.

The Vectorcardiographic Diagnosis of Myocardial Infarction

At each instant during cardiac systole all electric forces summate into one manifest force having magnitude, direction, and sense. This manifest force is called the cardiac vector and the projection of its terminus on various planes from instant to instant throughout systole traces the vectorcardiographic loop. Electrocardiographic leads, on the other hand,

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TABLE I

Case No.	Sex Age	Clinical Diagnosis	Vectorcardiographic Interpretation	Electrocardiographic Interpretation	Pathologic Data
1.	M 62	Coronary Heart Disease. Acute M. I. Pulmonary emphysema. Pulmonary Edema	10/22/51 Inferior, anterior, and septal M. I. R. and L. V. H. L O U* R A D†	10/23/51 Acute postero-lateral M. I.	10/26/51 Fresh post., and septal M. I. Anterior wall not sampled. L. and R. V. H. (4, 5, 6)**
2.	M 58	A. S. H. D. C. H. F. Diabetes Hemochromatosis Uremia	7/23/52 High post. and ant-lateral M. I. Acute Pericarditis L. and R. V. H. R A D L P D	7/22/52 Arborization block Acute antero-lateral M. I. Acute peri-carditis	7/24/52 Old ant., post, lateral M. I. Fresh ischemic necrosis apex. Diffuse fibrinous pericarditis. L. and R. V. H. (1, 2, 3, 4, 5, 7, 8)
3.	M 52	A. S. H. D. Old M. I. C. H. F. Low salt syndrome	10/4/51 Inferior, anterior, lateral M. I. L. and R. V. H. R A U L A D	10/4/51 Healed postero-lateral M. I.	11/6/51 Posterior, anterolateral, and septal M. I. L. and R. V. H. (2, 3, 5, 6)
4.	F 72	Coronary H. D. Acute M. I. Pulmonary infarct. A. F. C. H. F. Pulmonary emphysema	10/11/51 Inf., ant. M. I. R. and L. V. H. R A U L P D	10/11/51 Acute ant. and acute post. M. I.	11/7/51 Healing postero-septal and ant. M. I. L. and R. V. H. (7, 8, 9)
5.	M 77	A. S. H. D. A. P. A. F. Old M. I. ? Acute M. I. C. H. F. Pulmonary emphysema	12/11/51 Anterior and Septal M. I. L. V. H. L P D R P U	12/11/51 Anterior and septal M. I. I. V. Block	3/9/52 Old anterior and septal M. I. Also posterior M. I. Possible L and R. V. H. (4, 5, 6, 7, 8, 9)
6.	M 67	Hyp. and Cor. H. D. Old M. I. C. F. Asthma Chronic Bronchitis Emphysema. ? Pulm. Infarct.	1/22/52 Ant., Inf., Septal M. I. R. and L. V. H. L P U L P D	1/25/52 Antero-septal M. I. L. V. H.	5/15/53 Anteroseptal, posterior, apical, lateral fibrosis. L. and R. V. H. (5, 7, 8)
7.	M 62	A. S. H. D. Old and Acute M. I. C. H. F. Diabetes	12/17/51 Anterior, inferior, postero-lateral M. I. I. V. Block R A U L P D	12/17/51 Infero-lateral M. I. L. V. H.	3/7/52 Antero-lateral and posterior fibrosis R. and L. V. H. possible. (2, 3, 4, 5, 6, 7)
8.	F 57	Old M. I. C. H. F. Avitaminosis	1/6/54 Anterior, high postero-septal M. I. L. V. H. L A D R A D	1/8/54 Anterior M. I.	1/15/54 Anterior, posterior, and septal M. I. R. V. M. I. L. and R. V. H. (2)

are scalar derivatives of the cardiac vector, and are characterized by magnitude and sense only. The vectorcardiogram, therefore, permits an analysis of initial and early depolarization forces which is superior to the electrocardiogram. Except in situations in which the left ventricle is activated belatedly, as in left bundle branch block, or in an aberrant manner, as in anomalous atrioventricular excitation, these initial and early forces hold the clue to the diagnosis of infarction.[1, 4, 5]

Previous observations have shown that the initial and early depolarization forces have a characteristic orientation in various heart positions in the absence of localized myocardial disease.[6] The effect of the extrinsic

TABLE I

Case No.	Sex Age	Clinical Diagnosis	Vectorcardiographic Interpretation	Electrocardiographic Interpretation	Pathologic Data
9.	M 67	Coronary H. D. Angina Acute M. I. Aortic stenosis. Pulmonary emphysema. C. H. F. Diabetes	10/8/51 Posterior and septal M. I. R. and L. V. H. R. B. B. B. R P U R A D	10/16/51 Posterior, and acute extensive anterior M. I. L. V. H. R. B. B. B.	10/20/51 Anterior, posterior, and septal fibrosis L. and R. V. H. (Insufficient sampling of ant. wall.) (1, 5, 6)
10.	M 60	A. S. H. D. Old M. I. Acute M. I. Emphysema Old pulmonary infarct. C. H. F.	12/12/52 Anterior, posterior, septal M. I. L. V. H. L P U R P U	12/12/52 L. B. B. B.	2/24/53 Old posterior M. I. L. V. H. ? R. V. H. (4, 5, 6)

Age, sex, clinical diagnosis, vectorcardiographic and electrocardiographic interpretation, and pathologic findings. Date of taking the vectorcardiogram and electrocardiogram, and on which the autopsy was done, is given in the upper left hand corner of the appropriate box.

M.=Male F.=Female
M. I.=Myocardial infarction
A. S. H. D.=Arteriosclerotic heart disease
C. H. F.=Congestive heart failure
H. D.=Heart disease
A. F.=Auricular fibrillation
A. P.=Angina pectoris
R. and L. V. H.=Right and left ventricular hypertrophy
Post.=Posterior
Ant.=Anterior
Inf.=Inferior
I. V. Block=Intraventricular block
Hyp. and Cor. H. D.=Hypertensive and coronary heart disease
Pulm. Infarct=Pulmonary infarct
R. B. B. B.=Right bundle branch block
L. B. B. B.=Left bundle branch block

*Direction of initial forces } L.=Left R.=Right
†Direction of terminal forces } A.=Anterior P.=Posterior
D.=Down U.=Up

**Areas involved on posterior wall of left ventricle (2)
The numerals in the bottom line of each box in the column of Pathologic Data refer to involved areas (2).

factors, position and rotation, can be recognized, therefore, and distinguished from intrinsic factors, in the normal heart, in right and left ventricular hypertrophy, and in right and left bundle branch block. These forces represent the summation, from instant to instant, of the forces of depolarization of all the individual muscle fibers activated at the onset and early in ventricular systole. During this interval there are fewer fibers being activated than later when the entire ventricular mass is in the active state. The early part of the QRSsE loop, therefore, is less complex than the subsequent portions. When infarction occurs, or when myocardial fibers become irresponsive as the result of sub-lethal injury, the involved fibers are rendered electrically inert, and there is a new balance of forces. The new resultants are modified both as regards their spatial position and their magnitude; they tend to point away from the electrically inert areas. Although these changes may take place throughout the entire period of ventricular depolarization, modification of the early, less complex part of the curve is probably greater, and more easily recognized, than the alterations occurring later. The diagnostic changes, consequently, are more evident in the initial and early forces, than in the overall disposition of the loop.

It is necessary to have clearly in mind the spatial position of the initial and early forces of the normal vectorcardiogram, and alterations in these which are dependent exclusively on changes in position and rotation. The disposition of the terminal forces is sometimes of great help in determining heart position. Myocardial infarction is suspected if the orientation of the early forces cannot be accounted for entirely on the basis of extrinsic factors. Infarction of the interventricular septum is indicated by abnormal initial forces, and of the free wall of the left ventricle by abnormal early forces. Abnormal direction of inscription of the QRS loop is also diagnostic of free wall infarction.

If the QRSsE loop does not begin and end at the same point (O point), the loop is said to be open, and corresponds to S-T segment deviation in the electrocardiogram. In open loops the spatial relationship between the O point and the end of the loop is characteristic in acute anterior and posterior infarction, ventricular hypertrophy, and acute pericarditis. The T loops have an altered direction of inscription in infarction, and are long and narrow in acute, and small and round in healing and old infarction.

Description and Interpretation of Vectorcardiograms in Myocardial Infarction

Figure 1. The initial QRS forces are oriented to the left and up, and therefore, are abnormal, indicating disease of the interventricular septum. The early forces continue in an exaggerated posterior and upward direction, and are diagnostic of infarction of the anterior, and lower part of the posterior wall of the left ventricle. The QRS loop ends to the right, anteriorly, and inferiorly to the O point, resulting in an open QRSsE loop of the type seen in acute anterior and posterior infarction.

The clockwise inscription of the frontal, and counterclockwise inscription of the sagittal projections, is abnormal. This is frequently observed in posterior wall infarction, but also occurs in combined right and left ventricular hypertrophy.

Histologic correlation is excellent, and superior to that of the electrocardiogram (See Table I).

Figure 2. The initial QRS forces point to the right, anteriorly, and down, which is normal for a horizontally placed loop. However, the continued rightward, anterior, and downward orientation of the early forces is characteristic of a high postero-lateral lesion. Nevertheless, quite early

FIGURE 1A

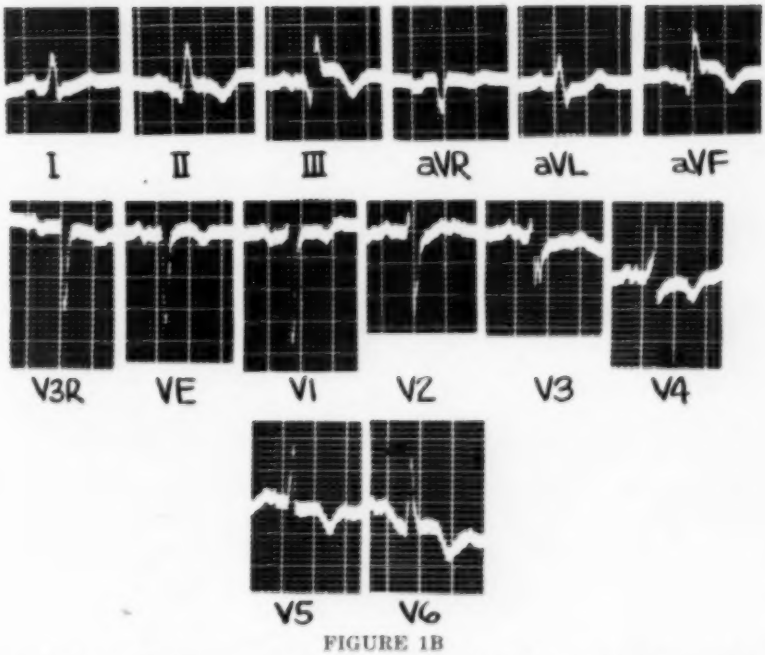

FIGURE 1B

Figure 1A: Vectorcardiogram in fresh posterior and septal myocardial infarction. Anterior wall not sampled. Left and right ventricular hypertrophy. In this and the following figures H= Horizontal, S = Sagittal, and F = Frontal plane projections, and arrows indicate direction of inscription of the QRS loop. See text and Table I.—*Figure 1B:* Electrocardiogram from same patient.

in the QRS interval there is pronounced posterior displacement, resulting in clockwise inscription of the horizontal projection, striking abnormalities which indicate anterior wall infarction.

The end of the QRS loop is to the left of, and anterior and inferior to the point of origin, a relationship which is characteristic of acute fibrinous pericarditis.

The clockwise inscription of the horizontally placed frontal loop, which

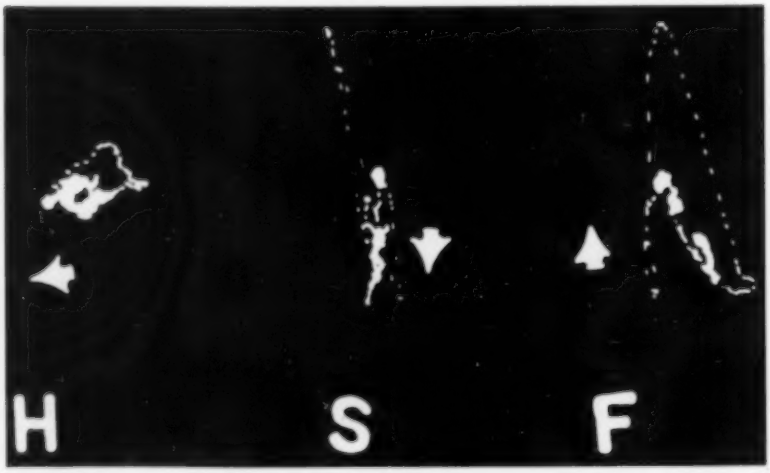

FIGURE 2A

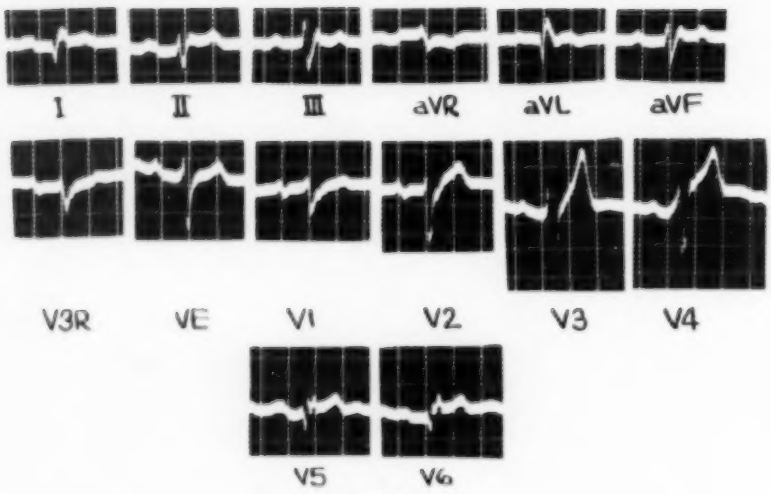

FIGURE 2B

Figure 2A: Vectorcardiogram in old anterior, posterior, and lateral wall infarction and diffuse fibrinous pericarditis (uremic). Fresh apical ischemic necrosis. Left and Right Ventricular Hypertrophy. See text and Table I.—*Figure 2B:* Electrocardiogram from same patient.

has features seen in left ventricular hypertrophy, is due to the infarct, or to right ventricular hypertrophy.

Histologic correlation is excellent, and greatly superior to that of the electrocardiogram.

Figure 3. There is no evidence of septal involvement, since the initial forces have a rightward, anterior, and barely upward orientation, which is normal in a horizontal loop. The early forces, however, continue far to the right, then swing sharply to the left, posteriorly, and upwards, denoting anterior, inferior, and lateral wall myocardial infarction. The QRS loop is closed and the T loops are rather small, indicating healed infarction.

The direction of inscription in each projection is the reverse of that expected in the normal, a common finding in infarction. However, clockwise inscription of a markedly horizontal frontal loop suggests right and left ventricular hypertrophy.

Histological correlation with the vectorcardiographic interpretation is good, and far superior to the electrocardiographic interpretation.

Figure 4. Septal involvement is excluded because the initial forces are normal (right, anterior, and barely superior). The early forces turn

FIGURE 3A

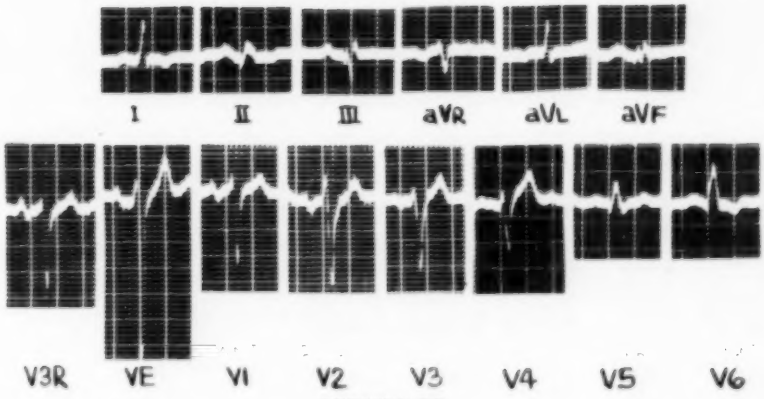

FIGURE 3B

Figure 3A: Vectorcardiogram in posterior, antero-lateral, and septal myocardial infarction. Left and Right Ventricular hypertrophy. See text and Table I.—*Figure 3B:* Electrocardiogram from same patient.

sharply posteriorly, superiorly, and to the left. From then on the loop remains posterior, superior, and to the left. These features are diagnostic of inferior and anterior myocardial infarction. The counterclockwise and clockwise inscription of the saggittal and frontal loops, respectively, occurs in infarction. However, clockwise inscription of a markedly horizontal frontal loop suggests right and left ventricular hypertrophy.

The QRS loops are only slightly open, and the T loops are small; the infarction is healing or old.

Correlation of the pathologic findings with the vectorcardiographic diagnosis is good. Only the septal involvement was missed. The electrocardiographic interpretation did not correlate as well; the infarction was thought to be acute, and the hypertrophy was not recognized.

Figure 5. The abnormal initial forces (left, posterior, and down), and the posterior displacement of the early forces, signify lesions in the interventricular septum, and in the anterior wall of the left ventricle, respectively.

The markedly horizontal position of the loop is strongly suggestive of left ventricular hypertrophy.

Correlation: The vectorcardiogram anticipated everything found at

FIGURE 4A

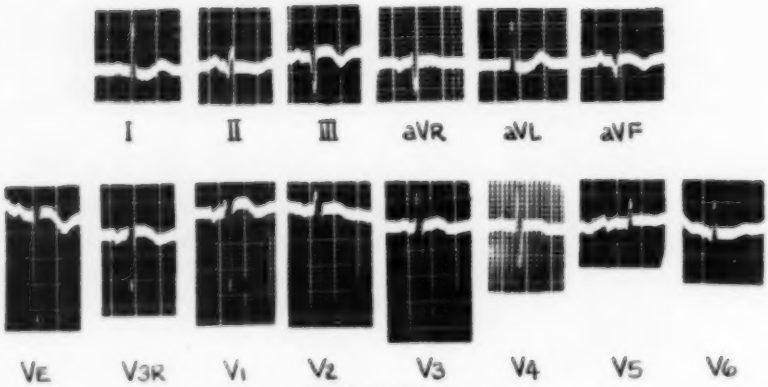

FIGURE 4B

Figure 4A: Vectorcardiogram in healing postero-septal and anterior myocardial infarction. Left and Right Ventricular Hypertrophy. See text and Table I.—*Figure 4B:* Electrocardiogram from same patient.

autopsy except the large posterior infarct. The posterior infarct and the left ventricular hypertrophy as well, were missed in the electrocardiogram.

Figure 6. The vectorcardiogram was interpreted as showing infarction of the anterior, inferior (diaphragmatic), and septal walls, and right and left ventricular hypertrophy. The initial forces (left, posterior, and up) call attention to the septal lesion, and the displacement of the early forces posteriorly and superiorly indicate anterior and inferior wall disease, respectively. The reversal in direction of inscription of all projections is common in infarction, but the clockwise inscription of the horizontally placed frontal loop suggests left and right ventricular hypertrophy.

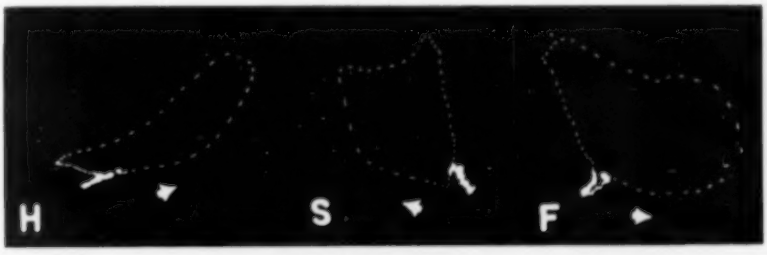

FIGURE 5A

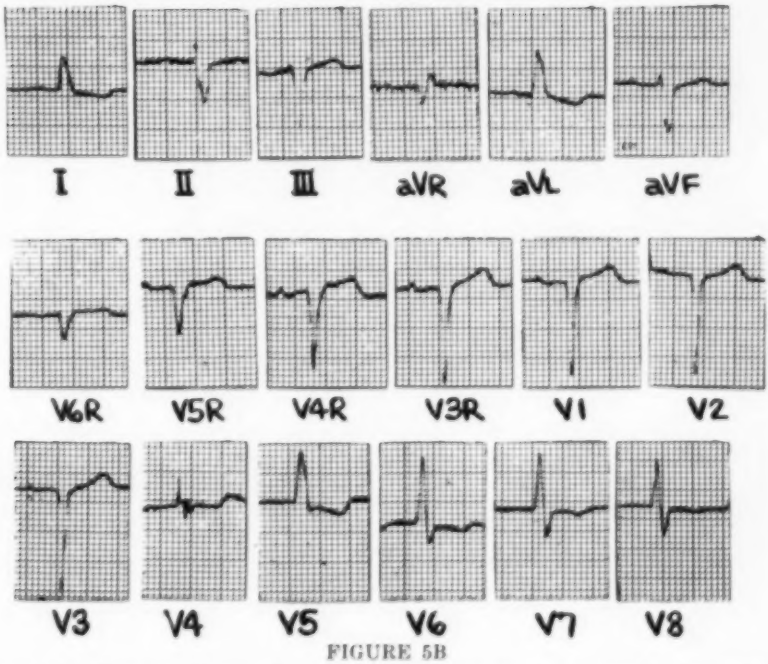

FIGURE 5B

Figure 5A: Vectorcardiogram in old anterior, posterior, and septal myocardial infarction. Possible left and right ventricular hypertrophy. See text and Table I.—*Figure 5B:* Electrocardiogram from same patient.

Correlation of the vectorcardiographic diagnosis with the autopsy findings is excellent, and greatly superior to the electrocardiogram.

Figure 7. Clockwise inscription of the posteriorly placed horizontal projection is characteristic of anterior wall infarction, and upward displacement of the early forces in a horizontally placed frontal loop is characteristic of an inferior lesion. Marked rightward displacement of the early forces seen in the horizontal and frontal projections indicates involvement of the lateral wall. The initial forces are normal (right, anterior, and barely up) and a diagnosis of septal infarction was not made.

The markedly horizontal position of the loop suggests left ventricular hypertrophy.

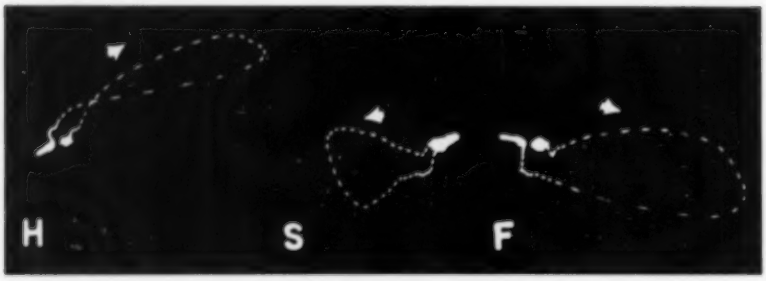

FIGURE 6A

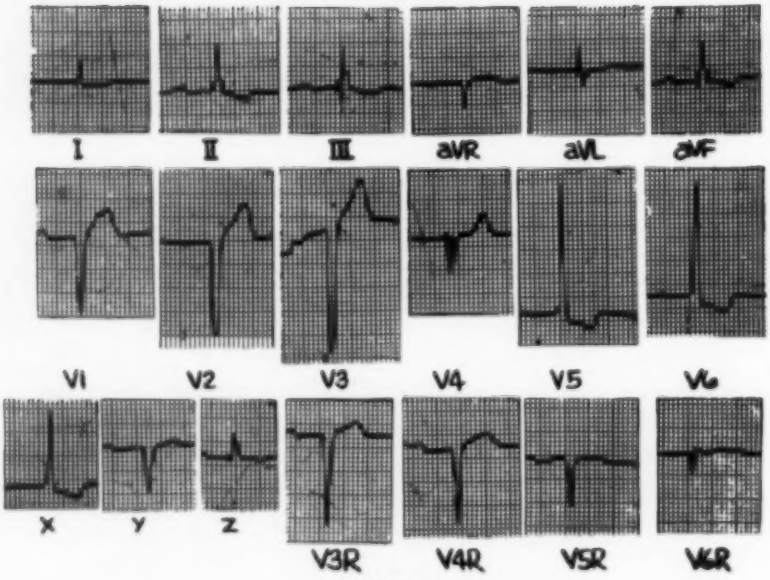

FIGURE 6B

Figure 6A: Vectorcardiogram in anteroseptal, posterior, apical and lateral wall fibrosis. Left and Right Ventricular Hypertrophy. See text and Table I.—*Figure 6B:* Electrocardiogram from same patient.

Correlation with the autopsy findings is excellent and superior to that of the electrocardiogram.

Figure 8. The vectorcardiogram was interpreted as showing an extensive myocardial infarct involving the postero-basal and septal myocardium, and the anterior wall. The abnormal initial forces (left, anterior, down) indicate a septal lesion, and continuation of the early forces in the same direction is diagnostic of postero-basal infarction. The almost completely posterior orientation of the spatial loop was considered evidence of extensive anterior myocardial infarction. The horizontally placed frontal projection suggests left ventricular hypertrophy.

Correlation with the autopsy findings is good, and greatly superior to the electrocardiogram.

Figure 9. The most obvious abnormality is clockwise inscription of the anteriorly placed horizontal projection, which is diagnostic of right ventricular hypertrophy, and the clockwise inscription of the horizontally placed frontal loop, which is characteristic of right and left ventricular hypertrophy. The terminal forces indicate right bundle branch block.

The abnormal initial forces (right, posterior, and up), and the abnormal upward displacement of the early forces, indicate septal and inferior myocardial infarction, respectively. The bizarre sagittal loop is due partly to rotation.

FIGURE 7A

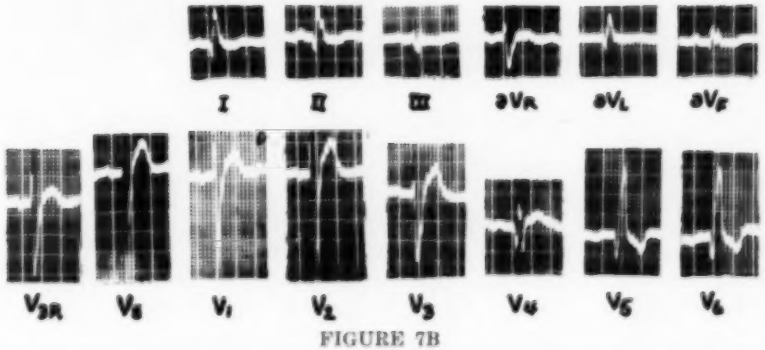

FIGURE 7B

Figure 7A: Vectorcardiogram in antero-lateral and posterior wall fibrosis. Right and Left Ventricular Hypertrophy possible. See text and Table I.—*Figure 7B:* Electrocardiogram from same patient.

Correlation with the autopsy findings is fair, and not as good as that of the electrocardiogram.

Figure 10. The abnormal initial and early forces (left, posterior, and up) indicate septal, inferior, and anterior wall infarction. The terminal forces are characteristic of left ventricular hypertrophy.

Correlation with the autopsy findings is excellent, and far superior to that of the electrocardiogram.

Discussion

The above observations clearly indicate that the vectorcardiogram is capable of revealing the presence of myocardial infarction. The cardiac vector is analyzed in relation to the three natural coordinate axes of the body as revealed by projection onto three mutually perpendicular planes, horizontal, sagittal, and frontal.[1] Except in instances of unusual heart position, displacement of electrical forces in the antero-posterior axis occurs in anterior and high posterior wall lesions, in the transverse axis in lateral wall and septal region, and in the vertical axis in lesions involving the inferior or basal portion of the posterior wall of the left ventricle. The abnormal vectors are oriented posteriorly and down in anterior infarction, anteriorly and down in high posterior infarction, up

FIGURE 8A

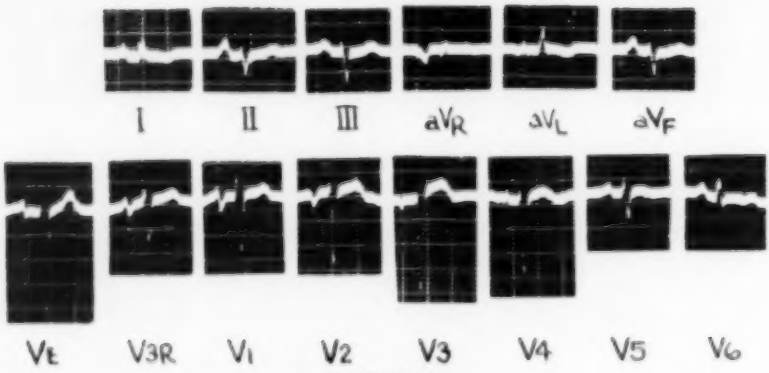

FIGURE 8B

Figure 8A: Vectorcardiogram in anterior, posterior, and septal myocardial infarction. Also infarction of right ventricular wall. Right and Left Ventricular Hypertrophy. See text and Table I.—*Figure 8B:* Electrocardiogram from same patient.

and posteriorly in infero-posterior wall lesions, to the left in lesions close to the interventricular septum, and to the right in lateral wall involvement.

Any departure from the normal orientation of the initial forces which cannot be explained by extrinsic factors, ventricular hypertrophy, or intraventricular block, indicates a septal lesion.

Acute infarction is characterized by an open QRS loop and long narrow T loops. In healing or old infarction the QRS loop tends to close and the T loop is small and round.[7] The manner in which the QRS loop is open is very helpful in differential interpretation. The end of the loop, in relation to the O point, is to the right, anterior, and up in acute anterior myocardial infarction; right, posterior, and down in posterior myocardial infarction; left, anterior, and down in acute fibrinous pericarditis; right and up, with no or minimal antero-posterior deviation in left ventricular

FIGURE 9A

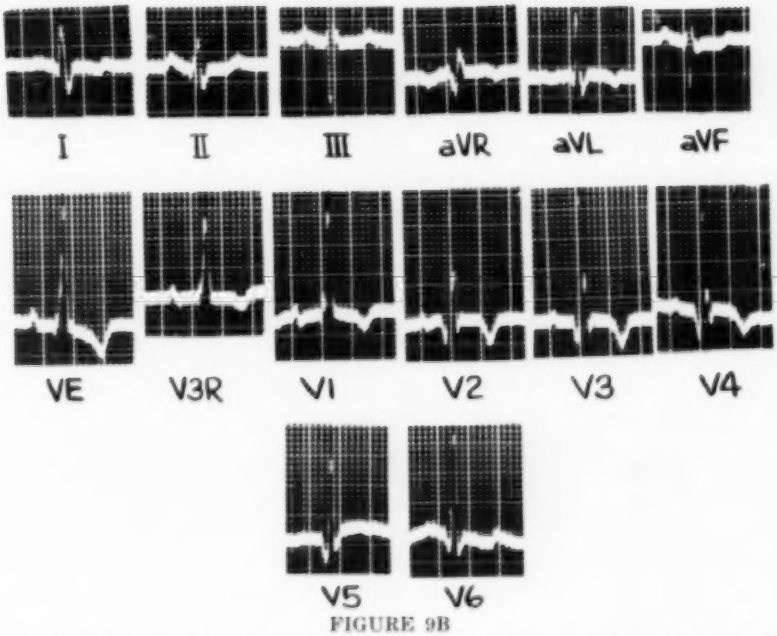

FIGURE 9B

Figure 9A: Vectorcardiogram in anterior, posterior, and septal fibrosis. Left and Right Ventricular Hypertrophy. See text and Table I.—*Figure 9B:* Electrocardiogram from same patient.

hypertrophy; and left, posterior, and up in right ventricular hypertrophy.

These criteria for the diagnosis of myocardial infarction apply to the reference system used in this laboratory.[1]

A comparison of the vectorcardiographic and electrocardiographic interpretations shown in Table I indicates superiority of the vectorcardiogram. The ten cases were chosen at random from a group of 50 cases studied histologically. Analysis of the entire autopsy series reveals an unqualified superiority of the vectorcardiogram over the electrocardiogram in disclosing myocardial infarction, as well as right and left ventricular hypertrophy, singly and in combination.[3] The results are summarized in Table II.

TABLE II

Vectorcardiographic and electrocardiographic diagnosis, and location at autopsy, of infarcted areas in 22 cases of myocardial infarction. Cases with Left Bundle Branch Block excluded.

Location of Infarct	No. Cases at Autopsy*	No. Diagnosed by Vectorcardiogram	Per Cent	No. Diagnosed by Electrocardiogram	Per Cent
Anterior	14	11	79.	8	57.
Posterior	21	16	76.	12	57.
Anterior and Posterior	13	10**	75.	4**	30.
Septum	17	11	65.	6	35.
Lateral	12	4	33.	3	25.
Anterior, Posterior, Lateral, and Septum	8	6†	75.	2†	25.
Any Location	22	18††	82.	17††	77.

*Cases with left bundle branch block excluded.
**Diagnosis of anterior and posterior myocardial infarction.
†Three or more areas diagnosed.
††Diagnosis of infarction, irrespective of its localization.

The bottom line in Table II is misleading, since it appears to indicate that there is no difference between the two methods of examination. However, the method of selection of patients is responsible for this, as in one experimental group only those cases were chosen for vectorcardiographic study whose electrocardiograms displayed the characteristic signs of infarct.* The overall results for the diagnosis of myocardial infarction are, therefore, heavily weighted in favor of the electrocardiogram. In spite of this, the data relating to localization and extent of infarction are obviously in favor of the vectorcardiogram. Furthermore, our total clinical experience, which does not have the authority of postmortem confirmation, has demonstrated the superiority of the vectorcardiogram in the diagnosis of infarction.

*Other groups included patients with hypertension, chronic pulmonary disease, rheumatic heart disease, congenital heart disease, and so on.

High posterior, and some anterior lesions are easily recognizable in the vectorcardiogram, but not in the electrocardiogram. Multiple infarcts appear to make the electrocardiogram more vulnerable, but do not impair the diagnostic usefulness of the vectorcardiogram. The vectorcardio-

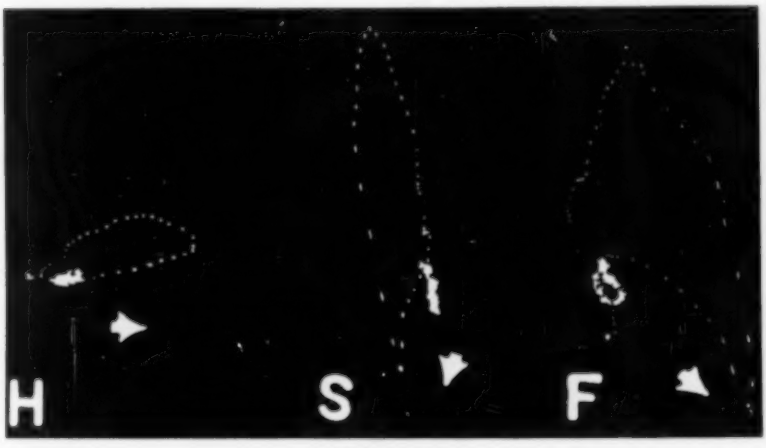

FIGURE 10A

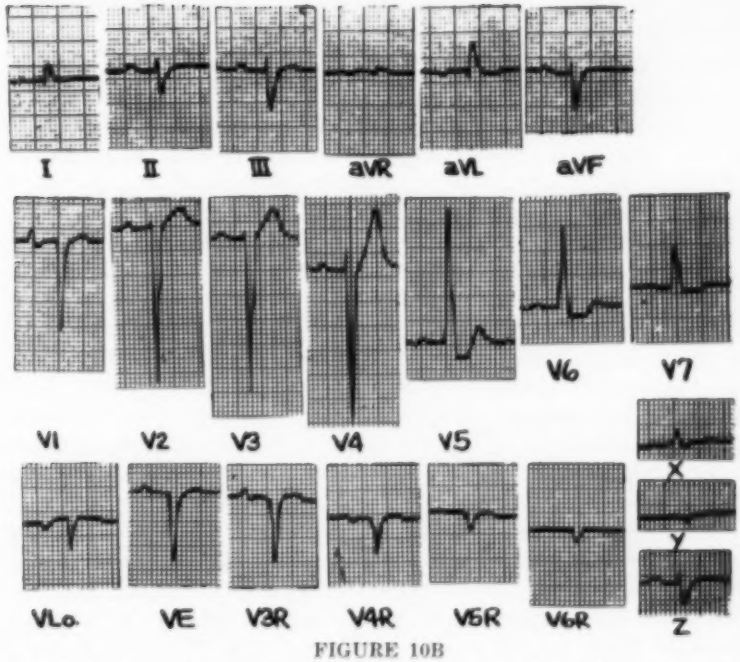

FIGURE 10B

Figure 10A: Vectorcardiogram in old posterior myocardial infarction. Left ventricular hypertrophy. Right ventricular hypertrophy. See text and Table I.—*Figure 10B:* Electrocardiogram from same patient.

gram does much better than the electrocardiogram in disclosing septal lesions, while both are poor indicators of lateral wall infarcts. A diagnosis of isolated septal infarction was made in only one case, by vectorcardiogram, but as in previous studies, the autopsy data indicate that free wall infarction always accompanies septal infarction.[2, 8] Similarly, isolated lateral wall infarction does not occur.

Right and left ventricular hypertrophy, singly and combined, can be recognized much more readily in the vectorcardiogram than in the electrocardiogram. This is even more strikingly apparent when hypertrophy and infarction coexist.

A false diagnosis of myocardial infarction is one of the serious pitfalls in the laboratory diagnosis of coronary heart disease. In the series referred to, however, a vectorcardiographic diagnosis of myocardial infarction was not made in a single instance in which autopsy failed to disclose such a lesion. On the other hand, one such electrocardiographic error was made. While not a single false vectorcardiographic diagnosis of myocardial infarction was made, over diagnosis occurred three times. In these three cases the diagnosis of anterior wall infarction was not confirmed at autopsy, although infarction existed elsewhere. A similar error in electrocardiographic diagnosis was made, also in three cases. In two additional cases a diagnosis of anterior infarction was made by vectorcardiogram and electrocardiogram but pathologic correlation was impossible as sections from the anterior wall were not available; myocardial infarction was present elsewhere in these two cases.

SUMMARY AND CONCLUSIONS

1. The initial, early, and terminal forces associated with ventricular depolarization can be studied in greater detail and more accurately by spatial vectorcardiography than by electrocardiography.

2. The initial, early, and terminal forces are stereotyped in the normal heart, right and left ventricular hypertrophy, and right and left bundle branch block, and are altered in a characteristic way by changes in heart position and rotation. The initial and/or terminal forces help in establishing the position and degree of rotation of the heart.

3. Changes in the initial forces which cannot be explained on the basis of heart position and rotation are diagnostic of septal disease.

4. Changes in the early forces indicate infarction of the free wall of the left ventricle. Localization of infarction is established in reference to the three natural coordinate axes of the body. The abnormal vectors are oriented posteriorly and down in anterior infarction, anteriorly and down in high posterior infarction, up and posteriorly in infero-posterior wall lesions, to the left in lesions close to the interventricular septum, and to the right in lateral wall involvement. Reversal in the direction of inscription of the vectorcardiographic projections frequently results from displacement of the forces, and furnishes valuable additional help in the diagnosis of infarction. Clockwise inscription of the posteriorly placed horizontal projection is diagnostic of anterior infarction, and clockwise

inscription of the horizontally placed frontal loop is diagnostic of posterior wall infarction.

5. The QRS loop is open in acute infarction, and the position of the end to the beginning of the loop is characteristic in anterior infarction, posterior infarction, acute pericarditis, and right and left ventricular hypertrophy. The T loop is long and narrow in acute, and small and round in healing or old myocardial infarction.

6. Comparison of vectorcardiographic and electrocardiographic interpretation in 50 cases studied at autopsy indicates unqualified superiority of the vectorcardiogram in the diagnosis of myocardial infarction, and right and left ventricular hypertrophy, singly or combined. The vectorcardiogram is superior for localization, and for detecting multiple areas of infarction. This superiority is evident in relation to anterior, posterior, and septal infarction. False vectorcardiographic diagnosis of infarction did not occur in this series.

7. Spatial vectorcardiography is a useful method of examination and its use should be extended.

RESUMEN Y CONCLUSIONES

1. Las fuerzas iniciales, precoces y terminales asociadas con la despolarización pueden estudiarse con mayor detalle y exactitud por la vectorcardiografía espacial que por la electrocardiografía.

2. Las fuerzas iniciales, tempranas y terminales están estereotipadas en el corazón normal, en la hipertrofia ventricular derecha e izquierda y en el bloqueo de ramas derecha e izquierda y son alteradas de manera característica por cambios en la posición del corazón y por larotación. Las fuerzas iniciales y/o terminales ayudan para establecer la posición y grado de rotación del corazón.

3. Los cambios en las fuerzas iniciales que no pueden explicarse sobre la base de la posición del corazón o la rotación son diagnósticos de enfermedad septal.

4. Los cambios en las fuerzas precozmente indican infarto de la pared libre del ventrículo izquierdo.

La localización del infaro se establece en relación a los tres ejer coordinados del cuerpo. Los vectores anormales se orientan posteriormente y hacia abajo en el infarto anterior; anteriormente y haciaabajo en el infarto posterior y hacia arriba zy posteriormente en las lesiones inferoposteriores de la pared; hacia la izquierda en las lesiones próximas al septum iterventricular y a la derecha en el caso de daño de la pared lateral. La inversión en la dirección de la inscripción de las proyecciones vectocardiográficas resultan a menudo del desplazamiento de las fuerzas y da valiosa información adicional en el diagnóstico del infarto. La inscripción en el sentido de las manecillas del reloj de la proyección posterior horizontalmente colocada hace diagnóstico de infar to anterior y la inscripción en el sentido del reloj del gancho frontal ubicado horizontalmente hace diagnóstico del infarto de pared posterior.

5. El gancho QRS es abierto en el infarto agudo y la posición y la

posición de su término al principio del gancho es característica en el infarto anterior, en el posterior, en la pericarditis aguda y en la hipertrofia derecha e iquierda ventriculares. La onda T es larga y estrecha en el infarto agudo y espequeña y redonda en el infarto en curación o antiguo.

6. La comparación de la vectocardiografía y la electrocardiografía en su interpretación en 50 casos estudiados a la autopsia indica una ni calcificada superioridad del vectocariograma en el diagnóstico del infarto del miocardio así como en el de la hipertrofia derecha é izquierda ventriculares, ya sea simple o combinadamente.

El vectocardiograma es superior para la lozalización y para descubrir áreas múltiples de infarto. Esta superioridad es evidente en relación a los infartos anterior, posterior y septal. En estas series no ocurrieron falsos diagnósticos vectocardiográficos de infarto.

7. La vectocardiografía espacial es un método útil para el examen y su uso debe ser más amplio.

RESUME

1. Les vecteurs initiaux, précoces et terminaux qui peuvent être créés par la dépolarisation ventriculaire peuvent être étudiés avec plus de détails et plus de précision que par l'électrocardiographie par le vectocardiographie spatiale.

2. Les vecteurs initiaux, précoces et terminaux sont stéréotypés dans le coeur normal, dans l'hypertrophie du ventricule droit et gauche, dans les blocs de branches droits et gauches, et sont modifiés d'une façon caractéristique lors de modifications de la position du coeur et lors de sa rotation. L'étude des vecteurs initiaux et terminaux peut aider à établir la position et le degré de rotation du coeur.

3. Les modifications des vecteurs initiaux qui ne peuvent être expliquées par des questions de position ou de rotation cardiaque permettent de diagnostiquer une lésion septale.

4. Les altérations des vecteurs précoces sont la preuve d'un infarctus de la paroi libre du ventricule gauche. La localisation de l'infarctus s'établit en fonction des trois coordonnées normales du corps. Les vecteurs anormaux sont orientés en arrière, et en bas, dans les infarctus antérieurs, en avant et en bas dans les infarctus postérieurs haut situés, en haut et en arrière dans les lésions de la paroi inféro-postérieure. Ils sont orientés vers la gauche dans les lésions qui sont proches du septum interventriculaire et vers la droite sur les altérations de la paroi latérale. Des inversions de la direction d'inscription de la projection vectocardiographique sont les résultats fréquents du déplacement des vecteurs et sont un auxiliaire précieux dans le diagnostic d'infarctus. La rotation horaire de la projection vectocardiographique horizontale, placée en arrière traduit le diagnostic d'infarctus antérieur et la rotation horaire de l'onde frontale placée horizontalement traduit le diagnostic d'infarctus de la paroi postérieure.

5. L'onde QRS est ouverte dans l'infarctus en phase aigue et sa position de la terminaison au commencement est caractéristique dans l'infarctus antérieur, l'infarctus postérieur, la péricardite aiguë, l'hypertrophie ventriculaire droite et gauche. L'onde T. est longue et étroite dans l'amélioration brusque, temporaire et rapide, ou dans un infarctus myocardique ancien.

6. La comparaison de l'interprétation vectocardiographique et électrocardiographique dans 50 cas étudiés á l'autopsie montre la remarquable supériorité du vectocardiogramme dans le diagnostic d'infarctus myocardique, d'hypertrophie ventriculaire droite et gauche, simple ou associée. Le vectocardiogramme est supérieur à l'électrocardiogramme pour diagnostiquer les localisations et les multiples zones d'infarctus ainsi qu'en ce qui concerne l'infartus antérieur, postérieur et septal. Dans la série de cas étudiés, il n'y ent pas d'erreur de diagnostic vectocardiographique pour l'infarctus.

7. La vectocardiographie spatiale est un procédé d'examen utile et dont l'emploi devrait être étendu.

REFERENCES

1 Wolff, L., Richman, J. L. and Soffe, A. M.: "Spatial Vectorcardiography Review and Critique," *N.E.J. Med.*, 248:810 and 851, 1953.

2 Wolff, L., Mathur, K. S. and Richman, J. L.: "The Diagnosis of Posterior Myocardial Infarction," *Am. Heart J.*, 46:21, 1953.

3 Soffe, A. M., Wolff, L., Richman, J. L., Reiner, L. and Mazzoleni, A.: "Comparative Value of Electrocardiographic and Vectorcardiographic Diagnosis; Pathologic Correlation," *Presented at the Second International Cardiological Congress*, Washington, D. C. September, 1954.

4 Richman, J. L. and Wolff, L.: "Left Bundle Block Masquerading as Right Bundle Branch Block," *Am. Heart J.*, 47:383, 1954.

5 Wolff, L. and Richman, J. L.: "The Diagnosis of Myocardial Infarction in Patients with Anomalous Atrioventricular Excitation (Wolff-Parkinson-White Syndrome)," *Am. Heart J.*, 45:545, 1953.

6 Wolff, L., Richman, J. L. and Soffe, A. M.: "The Effect of Heart Position and Rotation on the Cardiac Vector: An Experimental Study," *Am. Heart J.*, 47:161, 1954.

7 Duchosal, P. W. and Sulzer, R.: "La Vectorcardiographie," 172 pp. Basel: S. Karger, 1949.

8 Osher, H. and Wolff, L.: "The Diagnosis of Infarction of the Interventricular Septum," *Am. Heart J.*, 45:429, 1953.

The Ventricular Pulsations in Myocardial Infarction; a Fluoroscopic and Kymographic Study

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The study of left ventricular contraction by fluoroscopy,[1] roentkymography,[2, 3] and electrokymography[4-6] has proved to be of great practical value in the detection of myocardial disease, particularly myocardial infarction. Visualization and analysis of the left ventricular border movements should be part of every routine cardiac fluoroscopy, affording a simple and accurate method for detecting localized areas of myocardial disease which is readily available in the office and clinic. For more detailed study and analysis of left ventricular border pulsations than can be attained by fluoroscopy one can utilize the electrokymograph or roentgenkymograph which are available in the clinic and hospital.

Normal Left Ventricular Contraction

Fluoroscopic observation of the cardiac pulsations in normal individuals reveals uniform inward or medial movement of the entire left ventricular border during systole (Fig. 1). The timing of the ventricular border movements can be checked by comparison with those of the aortic knob. The latter segment moves laterally and expands during ventricular systole as a result of aortic filling. Thus, there is a seesaw movement of the left cardiac border about the middle segment in the region of the left auricular appendage ("point of no motion"). The upper arterial segment, comprising the pulmonary artery and aorta, expands during systole and moves outward and laterally. The left ventricular segment below it contracts during systole and moves inward or medially. Although the inward movement of all points along the left ventricular border is not entirely simultaneous it is fairly uniform. The upper part of the left ventricular border moves inward and downward, the middle or supra-apical part moves inward and medially and the lower or diaphragmatic segment moves inward and upward.

The left ventricular movements can be studied also in the slight left and right oblique views. In general, the character of the ventricular pulsations in the oblique views is similar to that of the P-A view. In the left oblique view, however, the movements may be rotatory or spiral in character and the inward or medial movement may be preceded by a transient outward expansile movement at the onset of systole. Electrokymography has demonstrated that these variations are due mainly to positional factors affecting the border movements.[5, 7]

Electrokymography is a graphic method for recording the cardiac pulsations[7, 8] and is based on the use of a photosensitive multiplier tube which is attached to the ordinary fluoroscopic screen. When the position of the tube is adjusted so that the slit lies over the heart border to be studied,

the movements of the border produce cyclic variations in the intensity of fluorescent light entering the photo-tube. This is transformed into a pulsating flow of electrons which is amplified and recorded as a phasic curve by a galvanometer, simultaneously with other reference tracings such as the carotid pulse and the heart sound tracings. The polarity of the apparatus is such that downward movement of the electrokymographic curve represents inward or medial movement of the underlying heart border, and upward movement of the curve represents outward or lateral movement of the underlying border.

The electrokymogram obtained from the left ventricular border has a characteristic appearance (Fig. 2). In general, it resembles a ventricular volume curve with superimposed positional movements at the onset of systole and diastole. A typical curve shows a steep downward limb during systole, representing inward or medial movement of the left ventricular border during the ejection phase. This may be preceded by a small transient upward movement lasting 0.02 to 0.06 second, which is generally due to a positional movement or change in shape of the heart at the onset of ejection. During the isometric phase preceding ejection, the curve may show a horizontal segment or a slight upward or downward movement

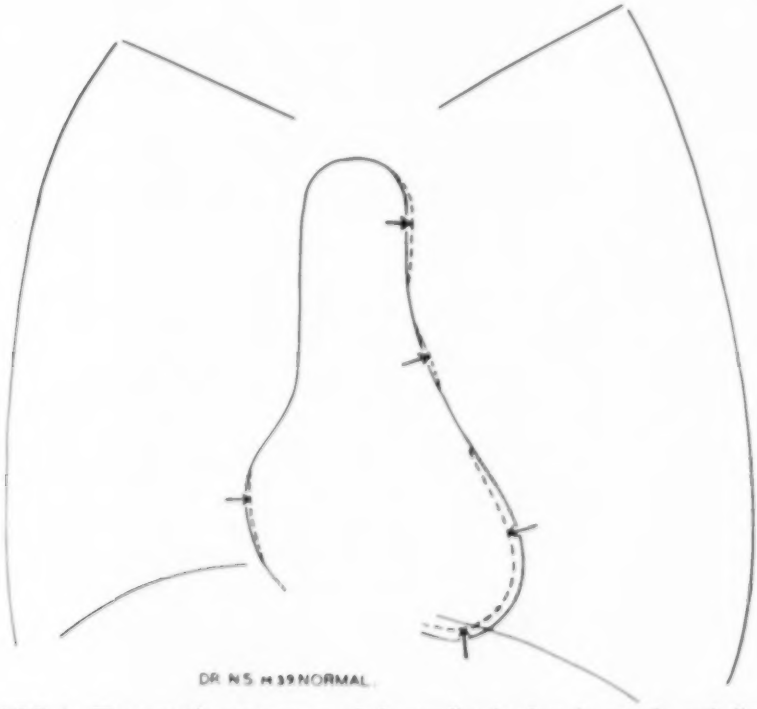

FIGURE 1: Fluoroscopic appearance of the cardiac borders in systole and diastole of a normal individual as traced from the fluoroscopic screen. The arrows indicate the direction of border movement during systole. The aortic and pulmonary artery segments move outward during systole while the left ventricle border moves inward.

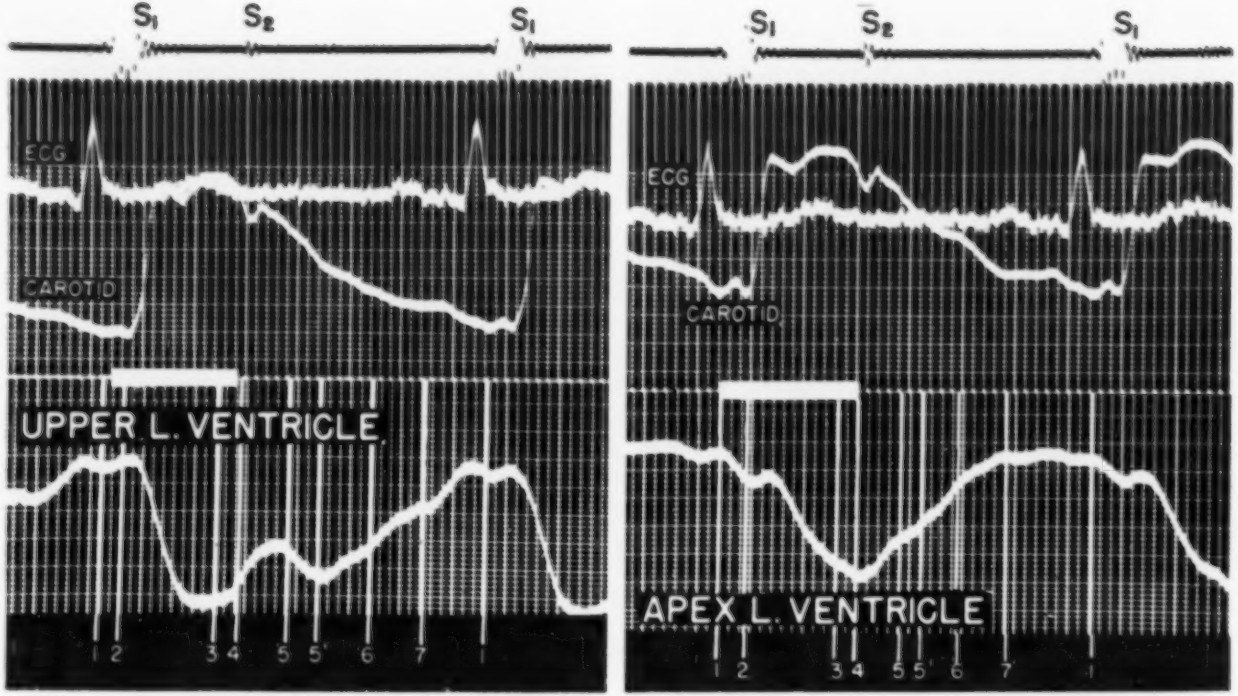

FIGURE 2: Normal Ventricular Electrokymograms. Electrocardiograms of the upper and apical segments of the left ventricular border of a normal individual, recorded simultaneously with the phonocardiogram, electrocardiogram and carotid pulse tracing. Downward and upward movements of the electrokymogram indicate inward and outward movements of the ventricular border, respectively. The ventricular curve may be subdivided into the following phases of the cardiac cycle: isometric contraction (1-2), ejection (2-3), protodiastole (3-4), isometric relaxation (4-5), rapid filling (5-6), slow filling (6-7), auricular contraction (7-1). The differences in configuration of the two curves are due to positional movements of the heart at the onset of systole and diastole.

lasting for 0.02 to 0.04 second. The steep downward movement of the ejection phase is succeeded by a V-shaped or horizontal segment, representing the isometric relaxation phase. This is then followed by upward movement of the curve during ventricular diastole and filling, which is fairly steep in its early portion (rapid inflow phase) and shallower in its late portion (slow inflow phase).

Ventricular Pulsation in Myocardial Infarction

Myocardial infarction produces characteristic abnormalities of left ventricular contraction which can be recognized visually during routine fluoroscopy[1] and recorded graphically by the roentgenkymograph[2, 3] and electrokymography.[4-6, 9] These abnormalities of left ventricular contraction can be detected during systole and diastole.

The abnormalities of the systolic phase may be classified in three main groups: 1. Localized diminution or absence of ventricular pulsation. 2. Partial reversal of pulsation during early systole. 3. Complete reversal of pulsation or paradoxical ventricular movement.

Localized Diminution or Absence of Ventricular Pulsation. In this abnormality there is observed during fluoroscopy a lack of movement localized to one segment of the left ventricular border (Fig. 3 and 4). This generally involves the lower half or apical region of the left ventricular border. Electrokymographic curves obtained from the involved segment

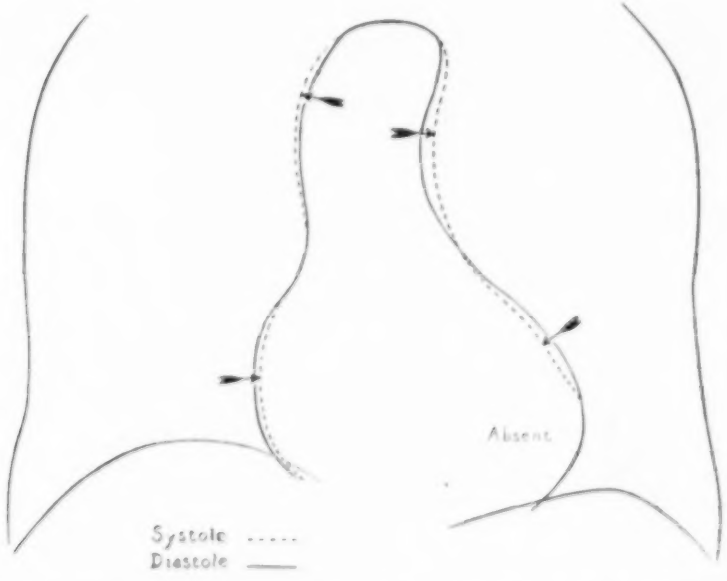

FIGURE 3: Localized absence of left ventricular pulsation observed fluoroscopically in a patient with previous myocardial infarction 4 years ago. During systole there is absence of inward movement of the lower third of the left ventricular border in contrast to normal pulsation of the upper left ventricular border and aorta.

show marked diminution in amplitude and even absence of the systolic and diastolic waves.

To be significant the diminished amplitude of pulsation should be localized to one segment of the left ventricular border rather than involve the entire cardiac border. Also, diminished movement of the left ventricular border just above the diaphragm in obese patients or of the apical segment when it is obscured by a pericardial fat pad should be interpreted with caution. This abnormality probably results from impairment of left ventricular contraction which is not severe enough to produce actual ballooning out of the ventricular wall or reversal of pulsation during systole.

Reversal of Left Ventricular Pulsation (Paradoxical Movement). In this abnormality the involved segment of the left ventricular border balloons out during systole. This may be a transient phenomenon at the onset of systole following which it is succeeded by a delayed inward movement (partial reversal of pulsation) or it may be sustained for the entire duration of systole (complete reversal of pulsation).

Fluoroscopically, reversed or paradoxical pulsation is visualized as an outward movement of a segment of the left ventricular border, generally the middle or apical segment (Fig. 5). As the uninvolved upper segment of the left ventricular border moves inward or medially during systole (timing checked with the aorta and pulmonary artery) the lower segment moves outward and laterally. Thus, there is a seesaw movement over

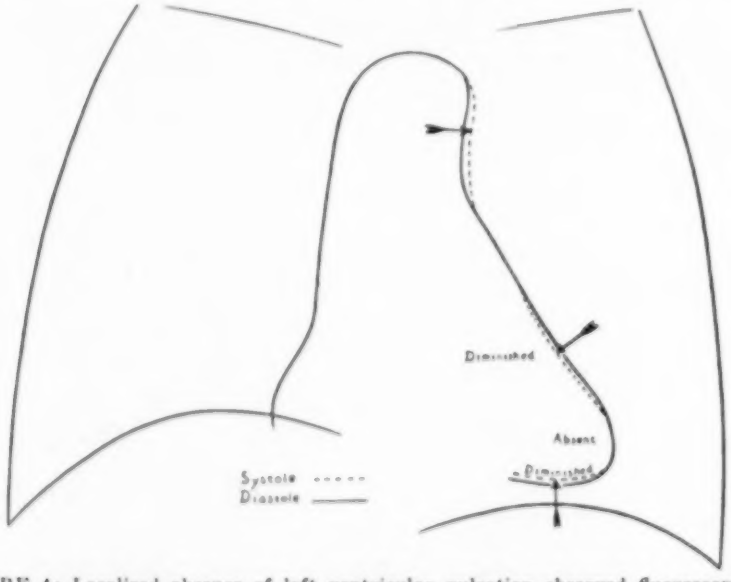

FIGURE 4: Localized absence of left ventricular pulsation observed fluoroscopically in a patient with previous myocardial infarction one year ago. The upper segment of the left ventricular border moves inward during systole and the inferior or diaphragmatic segment moves inward and upward. However, the apical segment fails to contract.

the left ventricular border. The involved weakened segment of the left ventricular border is ballooned out or actually thrust out as the remaining uninvolved border contracts and moves inward. In some cases there is a wavy motion over the left ventricular border and in other cases there may be a double pulsation during systole.

In the electrokymogram, reversed or paradoxical left ventricular pulsation is manifested by upward movement of the curve which generally begins during the isometric phase of systole or at the onset of the ejection phase. This is sustained for a variable period of time, depending on whether the reversal is partial or complete. In partial reversal, the upward systolic movement is replaced in early or mid systole by inward movement which may be normally steep or abnormally retarded (Figs. 6 and 7). The upward movement generally lasts for more than 0.08 second. This differentiates it from a positional movement which may occur in a normal heart but the duration of which is less than 0.08 second.

In complete reversal of pulsation the upward movement of the curve is sustained for the entire duration of systole and is followed by a collapse of the curve at the onset of diastole (Figs. 8 and 9). The latter generally occurs during or at the end of the isometric relaxation phase. The resulting curve is completely paradoxical and resembles an arterial curve instead of a normal ventricular curve (Fig. 10).

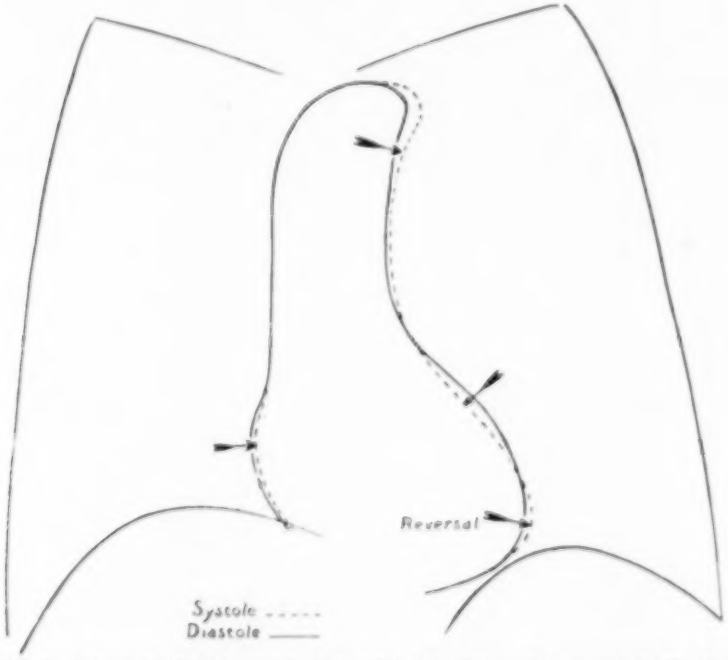

FIGURE 5: Reversal of left ventricular pulsation observed fluoroscopically in a patient with previous myocardial infarction three months ago. There is paradoxical pulsation of the lower half of the left ventricular border which moves outward during systole as the upper left ventricular border moves inward.

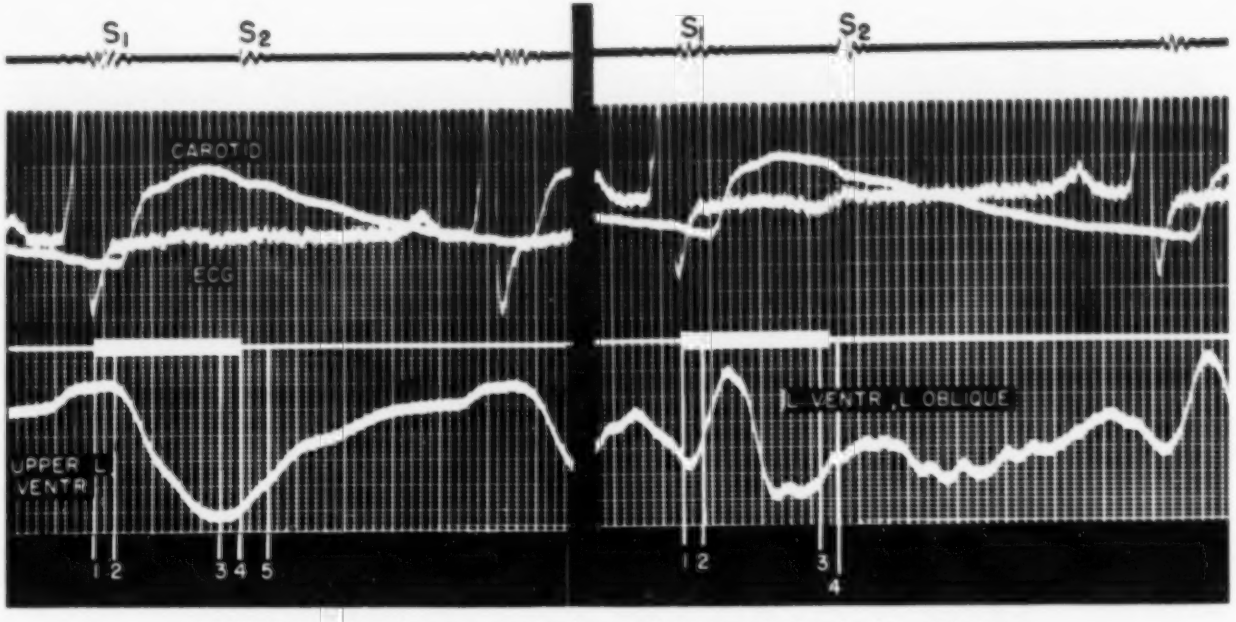

FIGURE 6: Electrokymograms showing partial reversal of pulsation of left ventricular border in a patient with previous myocardial infarction two years ago. The upper left ventricular border electrokymogram is normal. The curve recorded from the left ventricle in the left oblique view shows upward movement at the onset of systole which is sustained for approximately 0.12 sec. (each vertical line represents 0.02 sec.). There is marked diminution of pulsation during the remainder of the cardiac cycle. In this case the abnormal ventricular pulsation was observed and recorded only in the left oblique position. The ECG findings were obscured by bundle branch block.

Incidence of Abnormal Ventricular Contraction. Previous studies[1, 2, 9] of a large series of patients with proven myocardial infarction have indicated that partial or complete reversal of left ventricular pulsation occurs in approximately 50 to 60 per cent of patients, localized diminution or absence of pulsation in 20 to 25 per cent and normal pulsations in the remainder. These statistics were derived from fluoroscopic observation, electrokymography and roentgenkymography. Of course, the incidence of abnormal pulsation would be expected to be higher when the graphic methods are employed, owing to the objectivity of these methods, particularly electrokymography. For example, a slight reversal of pulsation may be missed during fluoroscopy but would be easily demonstrated when magnified approximately 20 times by the electrokymogram. On the other hand, a false positive diagnosis of reversed pulsation is occasionally made from fluoroscopy alone. In our experience, however, a well-trained fluoroscopist, who is aware of the abnormalities which occur following myocardial

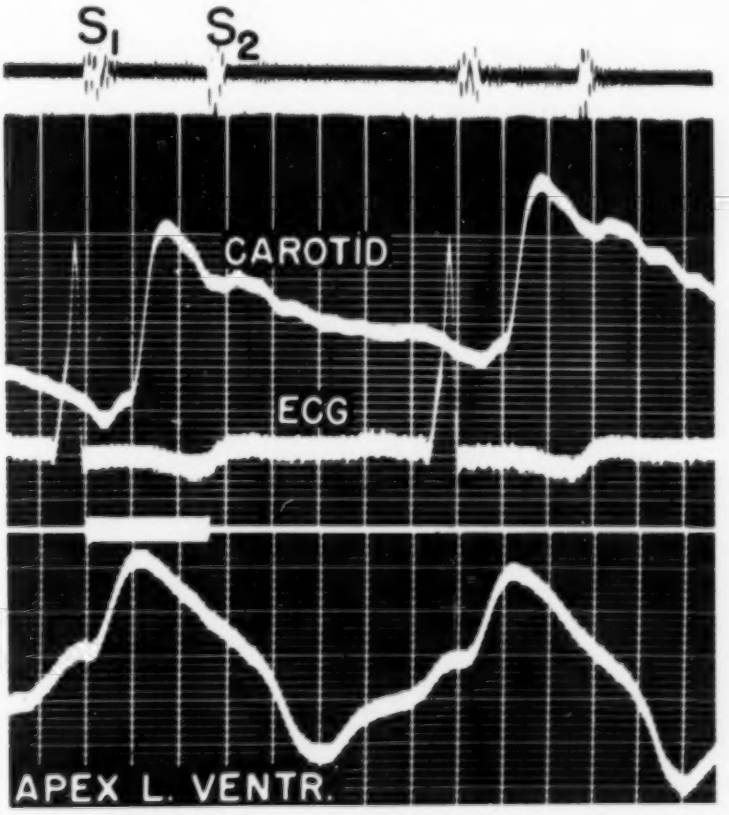

FIGURE 7: Electrokymogram of apex of left ventricular border in a 28 year-old man with aneurysmal dilatation of the left ventricle due to obscure congenital myocardial disease. The curve moves upward during early systole and shows delayed downward movement during late systole and early diastole. This is a partial reversal of pulsation and indicates impaired left ventricular contraction.

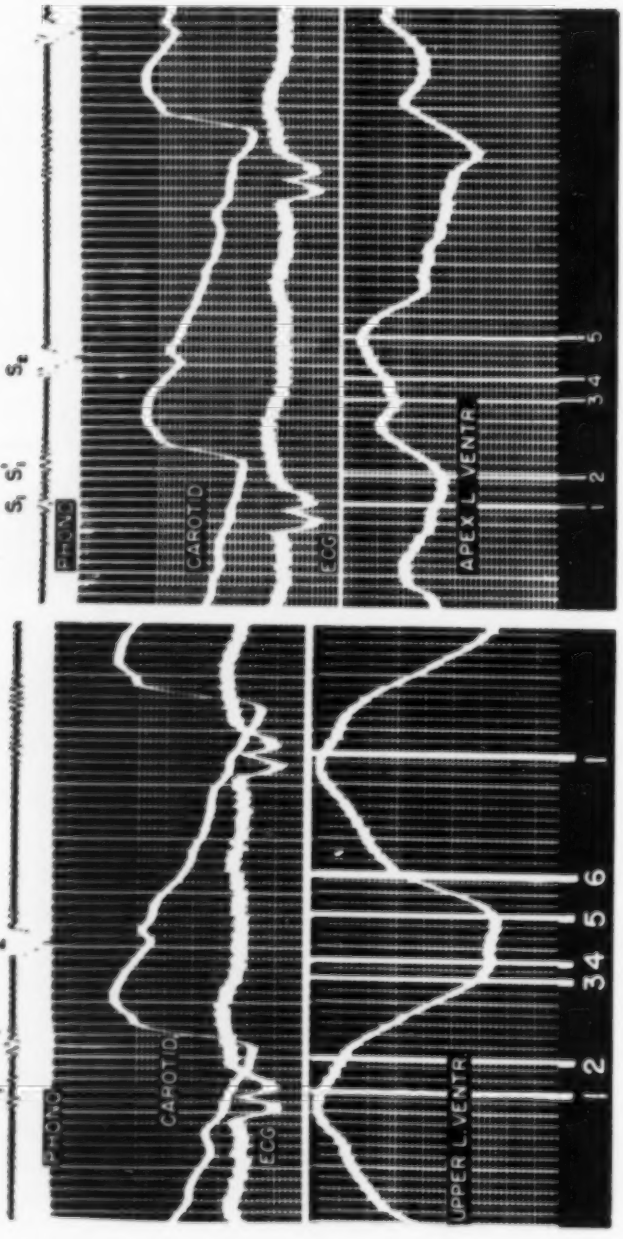

FIGURE 8: Electrokymograms showing complete reversal of left ventricular pulsation in a patient with previous anterior wall infarction three months ago. The upper left ventricular border movement is normal. The curve of the apical segment shows upward movement which is sustained for the entire duration of ventricular systole. This is a paradoxical curve and is pathognomonic of myocardial infarction.

infarction and who looks for them routinely, can become almost as accurate in visualizing the abnormal movements by fluoroscopy as with the graphic method. It is only when the ventricular movements are markedly diminished in amplitude, particularly in the presence of a markedly dilated heart, that the fluoroscopist may have difficulty in establishing the presence or absence of paradoxical movement in addition to the diminished movement. It is in this type of case that electrokymography attains its greatest practical clinical value as a diagnostic tool.

Location of Abnormal Ventricular Movements. The most frequent site of abnormal pulsation in myocardial infarction is the lower half or third of the left ventricular border. Not infrequently in a large infarct the reversal involves almost the entire ventricular border with only a small upper segment below the left auricular appendage which is uninvolved. On the other hand, reversed pulsation is occasionally limited to the middle or upper segment of the left ventricular border, with normal pulsation below this area (Fig. 10). In other cases the reversal is limited to the apical region just above the diaphragm. In sthenic individuals or in the presence of left ventricular hypertrophy the apex may be covered by the diaphragm and the abnormal ventricular pulsation is exposed only during a deep inspiration. In such a case one must guard against the patient straining while he holds his breath since he may perform the Valsalva maneuver which may result either in marked diminution in amplitude of pulsation or occasionally in reversed pulsation even in the absence of heart disease.

In the majority of cases the abnormal left ventricular pulsations are visualized or recorded in the P-A view. This holds true regardless of the location of the infarction as determined by the electrocardiogram.[8] In other words, whether the infarction is located on the anterior, lateral or diaphragmatic surface of the left ventricle, the abnormal movement is observed in the P-A view. Not infrequently, however, rotating the patient into a very slight left or right oblique view (5 to 15°) may accentuate the abnormal border movement (Fig. 6). Thus, in anterior infarction reversal of pulsation may be more readily visualized at the apex when the patient is rotated very slightly into the right anterior oblique rotation. In posterior infarction reversed pulsation may be visualized in the slight left oblique view as well as in the P-A view. It is only in the occasional case of posterior or diaphragmatic wall infarction that reversed pulsation is observed only in the left oblique view and not in the P-A view.

Onset and Duration of Abnormal Ventricular Pulsation. It is difficult to ascertain the exact time of appearance of absent or reversed ventricular pulsation following clinical myocardial infarction since patients with acute infarction are not generally fluoroscoped or examined kymographically during the acute stage. However, our studies[2] with serial roentgenkymograms have suggested that the abnormal pulsation is often present during the first week following the clinical attack and almost always has developed by the second week of the attack. Not infrequently it begins as

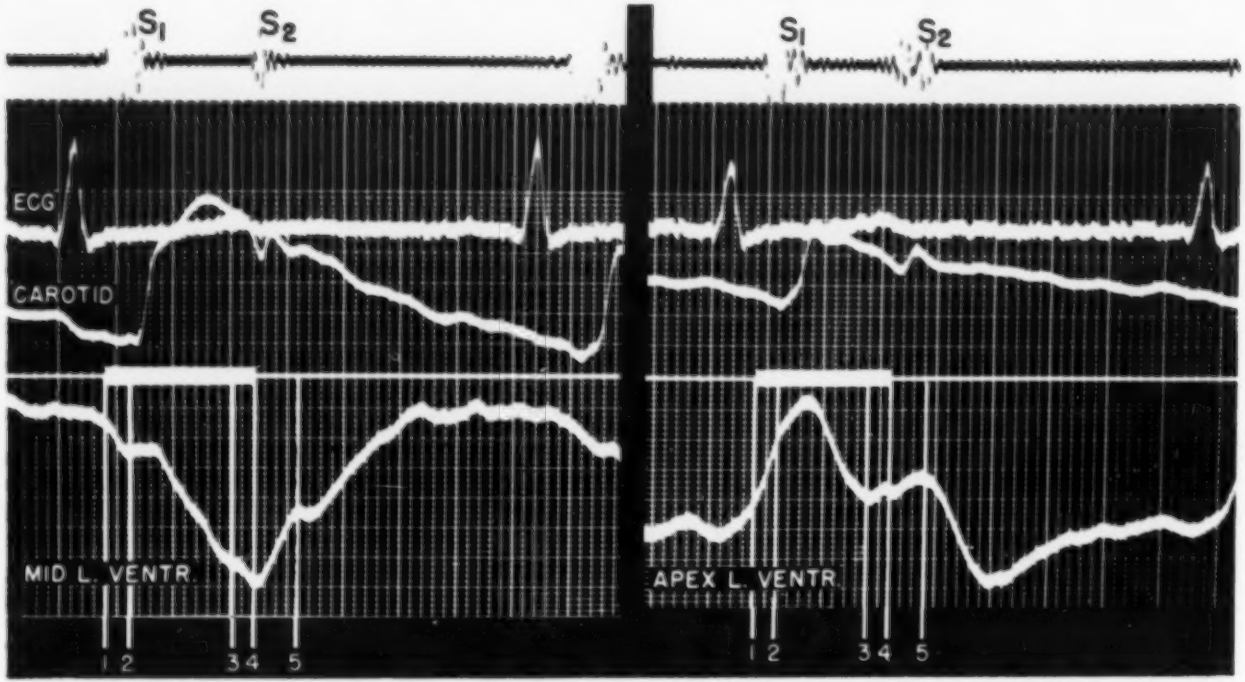

FIGURE 9: Electrokymograms of left ventricular border showing almost complete reversal of pulsation in a patient with previous anterolateral myocardial infarction four years ago. The curve obtained from the mid left ventricular segment is fairly normal. At the apex the curve is paradoxical showing marked upward movement which is sustained during the first half of systole. The curve shows a further fall at the end of systole and almost complete absence of filling movement during diastole.

a localized diminution or absence of pulsation which progresses to partial or complete reversal of pulsation.

Absent or reversed ventricular pulsation is often permanent. In a small percentage of cases it may disappear entirely or become less marked. In the great majority of cases, however, it lasts for many years. This may be of great diagnostic importance since the abnormal pulsation may persist even when the patient makes a complete clinical recovery and even if the electrocardiogram becomes normal or if the typical findings of myocardial infarction disappear.[3] In such a case, reversal of pulsation, visu-

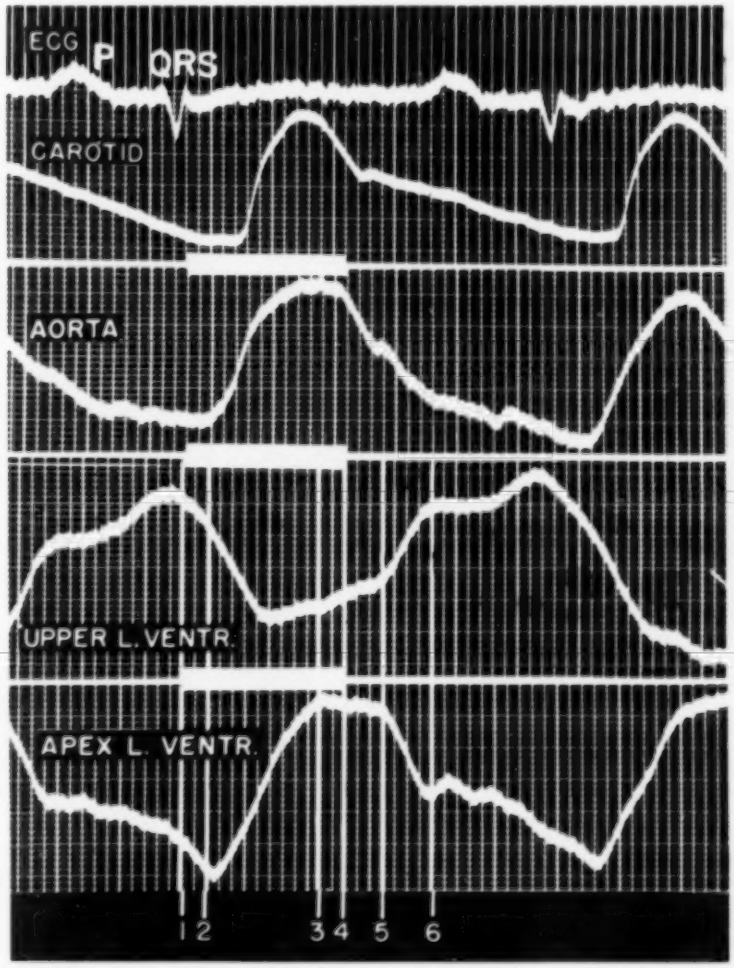

FIGURE 10: Electrokymograms of left ventricular border of patient with aneurysm of the left ventricle due to old myocardial infarction. The curve of the upper left ventricular border is normal. The curve of the apical segment is completely paradoxical, resembling the arterial curve of the aorta. There is upward movement of the curve which is sustained throughout systole and then falls in early diastole (5-6).

alized by fluoroscopy or recorded kymographically, may be the only residual finding indicative of previous myocardial infarction.

Differential Diagnosis of Reversal of Pulsation. As already stated, reversal of pulsation, particularly the partial type which is of short duration and limited to the early phase of systole (Fig. 6), must be differentiated from the transient outward movement of the left ventricular border observed in normal individuals which is of positional origin (Fig. 2). The latter is generally short in duration and rarely exceeds 0.06 second. Early reversal of pulsation lasts for at least 0.08 second and generally exceeds this duration.

Fluoroscopically, it may be difficult to differentiate early reversal of pulsation from a physiological outward movement. This may explain the occasional erroneous fluoroscopic diagnosis of myocardial infarction in an apparently normal individual. If the transient outward movement is observed over the entire left ventricular border the probability exists that it represents a physiological outward movement. Electrokymography, of course, may be the only accurate method to make this important differentiation.

The occurrence of an early physiological outward movement of the left ventricular border may be the result of several factors.[9] It may be due to a traction movement of the heart on the great vessels at the onset of contraction which produces a positional movement of the heart borders superimposed on the contractile movements of the various chambers. Secondly, it may be due to a change in the shape of the heart which has been demonstrated to occur in the early part of systole. This is caused by early unopposed contraction of the intraventricular septum before contraction of the free wall of the ventricle, leading to shortening of the heart in its longitudinal axis and transitory bulging outward of the free walls. This is cut short when the free walls contract and the heart borders move inward.

Physiologic Basis for Reversed Pulsation. Since the experiments of Tennant and Wiggers,[10] it is well known that following ligation of a coronary artery in the dog the ischemic portion of the myocardium ceases to contract within one minute and then begins to balloon out as the remainder of the ventricle contracts. The rise in intraventricular pressure at the onset of systole expands the weakened wall of the ischemic ventricular segment which collapses at the end of systole when the intraventricular pressure falls.

There is a close similarity of these events following experimental coronary ligation to those observed following clinical coronary occlusion except that it may take several days for the abnormal contraction to appear. Paradoxical left ventricular movement following myocardial infarction indicates the presence of a weakened area or scar in the ventricular myocardium which either fails to contract and remains motionless during systole or is ballooned out by the rising intraventricular pressure.

Several factors are responsible for the magnitude, extent and persistence

of the abnormal ventricular movement. These include the size and location of the infarcted area, the degree of healing and contraction of the resulting scar and the state of the collateral circulation. Experimental ballooning of the left ventricular wall following coronary ligation may be temporary or may be abolished by the release of the ligature or it may disappear spontaneously if the ligated vessel and resultant ischemic area are small.[10, 11] It may be increased in extent or magnitude by reducing the blood pressure to shock levels whereas restoration of normal blood pressure and reduction of coronary insufficiency results in complete or partial disappearance of the myocardial ballooning.[12]

From a physiologic standpoint every myocardial area of infarction associated with reversed or paradoxical pulsation acts dynamically as a ventricular aneurysm, despite the fact that radiological examination of the heart usually fails to show any localized bulge of the left ventricular border. Since the ballooning out of the ventricular wall occurs only in systole, it cannot be demonstrated by the conventional roentgenogram unless it is exposed only during systole, but has been strikingly demonstrated by cinematography of the heart. By this method a localized bulge of the left ventricular border is visible only in the frames exposed during ventricular systole and it disappears during diastole. When a typical aneurysmal bulge is visible on the roentgenogram, fluoroscopic examination or electrokymography will almost always demonstrate complete absence or reversal of ventricular pulsation over the involved segment as in any infarcted area of large extent (Fig. 10).

If the explanation is correct that reversed pulsation is due to ballooning of a weakened segment of the left ventricular wall, it is difficult to explain the frequent lack of correlation of the location of the abnormal movement and the actual site of infarction. It has been mentioned previously that irrespective of the actual site of infarction, the most common location of the abnormal ventricular movement is the apical segment of the left ventricular border in the P-A view, which may be at some distance from the infarcted area. The explanation for this is unknown. In previous publications[1, 2] it was speculated that it may be related to the spiral arrangement of the cardiac muscle bundles which wind around the apex of the heart from the base and are attached into the valve rings. It is possible that the muscle bundles act as a fulcrum; the weakening of any portion of this fulcrum whether anterior or posterior may result in ballooning out of the apical region.

SUMMARY

Cardiac fluoroscopy and kymography give important information in the study of the patient with myocardial infarction. The observation of marked localized diminution of pulsation of the left ventricular border or of complete or partial reversal of pulsation is indicative of a circumscribed area of myocardial damage, usually as a result of previous myocardial infarction. Such findings may be observed in the absence of other typical clinical findings of previous myocardial infarction. Careful exami-

nation of left ventricular border motion should be a routine part of every chest fluoroscopy.

The incidence, location, duration, clinical significance, differential diagnosis and physiologic basis for abnormal left ventricular motion in myocardial infarction is reviewed.

RESUMEN

La fluoroscopía y la kimografía cardiacas dan información importante para el estudio del enfermo de infarto miocárdico. La observación deuna marcada disminución de la pulsatilidad en área localizada del borde del ventrículo izquierdo o bien la observación de una inversión completa o parcial de esa pulsatilidad, indican una área circunscrita de daño del miocardio, generalmente como resultado de un infarto previo. El examen cuidadoso de la motilidad del borde ventricular izquierdo, debe ser una parte rutinaria de toda fluoroscopía del tórax. Se hace una revisión de la incidencia, ubicación, duración, signigcación clínica, diagnóstico diferencial y bases fisiológivas de la motilidad anormal del ventrículo izquierdo en el infarto del miocardio.

RESUME

La radioscopie du coeur et la kymographie apportent des renseignements importants pour l'étude des malades atteints d'infarctus du myocarde. La constatation d'une zone oú les pulsations sont nettement moins marquées sur le bord du ventricule gauche, ou la perception d'inversion completè ou partielle des pulsations, indique qu'il existe une zone circonscrite de lésions myocardiques. Dans ce cas, il s'agit généralement de la conséquence d'un infarctus myocardique antérieur.

De telles constatations peuvent être faites alors qu'il n'y a aucun autre signe clinique manifeste d'un infarctus myocardique antérieur. L'examen attentif des mouvements du ventricule gauche devrait être un élément de routine lors de tout examen radioscopique du thorax.

L'auteur étudie la fréquence, la localisation, la durée, la valeur clinique, le diagnostic différentiel et les bases physiologiques des mouvements anormaux du ventricule gauche dans l'infarctus du myocarde.

REFERENCES

1 Master, A. M., Gubner, R., Dack, S. and Jaffe, H. L.: "The diagnosis of coronary occlusion and myocardial infarction by fluoroscopic examination," *Am. Heart J.*, 20:475, 1940.

2 Sussman, M. L., Dack, S. and Master, A. M.: "The roentgenkymogram in myocardial infarction. I. The abnormalities in left ventricular contraction," *Am. Heart J.*, 19:453, 1940.

3 Dack, S., Sussman, M. L. and Master, A. M.: "The roentgenkymogram in myocardial infarction. II. Clinical and electrocardiographic correlation," *Am. Heart J.*, 19:464, 1940.

4 Sussman, M. L., Dack, S. and Paley, D. H.: "Some clinical application of electrokymography," *Radiology*, 53:500, 1949.

5 Dack, S., Paley, D. H. and Sussman, M. L.: "A comparison of electrokymography and roentgenkymography in the study of myocardial infarction," *Circulation*, 1:551, 1950.

6 Schwedel, J. B., Samet, P. and Mednick, H.: "Electrokymographic studies of abnormal left ventricular pulsations," *Am. Heart J.*, 40:410, 1950.

7 Boone, B. R., Chamberlain, W. E., Gillick, F. G., Henny, G. C. and Oppenheimer, M. J.: "Interpreting the electrokymogram of heart and great vessel motion," *Am. Heart J.*, 34:560, 1947.
8 Luisada, A. A., Fleischner, F. G. and Rappaport, M. B.: "Fluorocardiography (electrokymography). II. Observations on normal subjects," *Am. Heart J.*, 35: 348, 1948.
9 Dack, S. and Paley, D. H.: "Electrokymography. I. The Ventricular Electrokymogram," *Am. J. Med.*, 12:331, 1952.
10 Tenant, R. and Wiggers, C. J.: "The effect of coronary occlusion on myocardial contraction," *Am. J. Physiol.*, 112:351, 1935.
11 Prinzmetal, M., Schwartz, L. L., Corday, E., Spritzler, R. J., Bergman, H. C. and Kruger, H. E.: "Studies on the coronary circulation. VI. Loss of myocardial contractility after coronary artery occlusion," *Ann. Int. Med.*, 31:429, 1949.
12 Corday, E., Bergman, H. C., Schwartz, L. L., Spritzler, R. J.and Prinzmetal, M.: "Studies on the coronary circulation. IV. The effect of shock on the heart and its treatment," *Am. Heart J.*, 37:560, 1949.

Remarks on the Graphic Diagnosis of Coronary Disease

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I. Typical and Atypical Electrocardiograms in Myocardial Infarction Caused by Acute Coronary Occlusion and Coronary Insufficiency

The electrocardiogram has been epic making in the diagnosis of coronary occlusion and is the main reason for the ever increasing frequency with which this disease is detected. The ordinary electrocardiographic diagnosis has become so well-recognized that it is now well to talk, not only of the typical electrocardiogram, but of the atypical electrocardiogram in myocardial infarction. It is time also to divide the study of myocardial infarction into two types, (1) the subendocardial type, secondary to coronary insufficiency, that is, without complete occlusion of a coronary artery, (2) the through-and-through type caused by coronary occlusion, that is, complete obstruction of a coronary artery. Coronary insufficiency is usually associated with coronary sclerosis but may be observed in a normal heart, for example, following hemorrhage. In coronary insufficiency the electrocardiogram shows RS-T depression, T-wave inversion, or RS-T depression and T-wave inversion combined, since the damage is localized in the subendocardial region. For the same reason, the outlook is much better in coronary insufficiency. The electrocardiographic alterations are usually transient, lasting a day or two, or one or two weeks or months; rarely they are permanent. The T-wave inversions are not usually as deep as those seen in coronary occlusion. There are no Q-waves or RS-T segment elevations because there is no through-and-through infarction. It may take a few days for the electrocardiographic changes to develop; hence, if an acute coronary episode of any kind is suspected the electrocardiogram should be repeated several times.

The type of electrocardiogram just described, that is, with RS-T depressions and T-wave inversions, but without deep Q-waves, is also seen in the premonitory phase of a coronary occlusion. Obviously there is a partial occlusion which causes coronary insufficiency. I believe that, once the artery is completely occluded, the clinical and electrocardiographic picture of coronary occlusion appears, that is, RS-T elevation is seen immediately. Within the first 12 to 24 hours the characteristic deep Q-wave is not seen but one can almost discern it if he looks intently. After 12 to 24 hours it is usually quite obvious. In anterior wall infarction the deep Q and RS-T elevations appear in leads I, aVL and in chest leads. In diaphragmatic or inferior wall infarction these alterations appear in leads II, III and aVF. In true "posterior" infarction, tall R-waves appear on the right side of the chest, i. e., in V_1 and $_2$. Q-waves may be seen in $V_{5, 6, 7}$ or V_8.

Doctor Jaffe has been in the forefront in the description of atypical changes in coronary occlusion. For example, in serial electrocardiograms, temporary improvement may occur.

I personally feel that after complete occlusion the electrocardiogram will show the typical Q and RS-T patterns in 95 to 97 per cent of the cases. It does seem that, rarely, deep T-waves alone are present. In these cases there is enough normal muscle in the subepicardium to prevent the appearance of Q-waves.

II. Cardiac Diagnostic Methods in Coronary Artery Disease: Electrocardiogram, 2-Step Exercise Test and Ballistocardiogram

The *"2-step" exercise electrocardiogram* has become a diagnostic means for demonstrating coronary disease in the presence of a normal resting electrocardiogram; if the resting electrocardiogram is abnormal there is, of course, no need for performing the "2-step" test. My co-workers and I feel strongly that in the presence of definite coronary disease, the "2-step" test, either the single or the double, is positive in 95 per cent of the cases. Also, when the *double* "2-step" test is negative, coronary disease is excluded in 95 per cent of the cases; of course not in every case. However, when one speaks of a positive "2-step" test alone, without knowledge of the clinical story, one is in an insecure position. A positive "2-step" test should not be given any significance unless the clinical story is known. There is a small percentage of normal people who show positive tests, the so-called "false" positives, and also many people with an anxiety state do this. Hence, to diagnose "coronary" disease on a positive "2-step" electrocardiogram without clinical data or correlation, is not sound.

Another point that must be mentioned is the need for standardization in the performance of the Master "2-step." The exertion must be standardized for age and weight. The heavier a person, the more work he does and the fewer trips are required. The older the patient, the less stamina he has and again fewer trips will be "normally" made. The test does not depend on previous training—it depends on age, weight and the state of the coronary circulation.

There have been numerous "modifications" of the Master "2-step" test. If the patient is walked until pain appears, if he is asked to "run up" a flight of stairs, if he squats and stands alternately, if he hops on one foot, or any other type of exercise is used, it is not the test that my colleagues and I have described. For example, every "accident" following an exercise test that we have investigated occurred after some test other than the "2-step" test. We have been fortunate, indeed, that in a score of thousands of cases under the supervision of my colleagues and myself, we have never had an accident. First, the test should never be done unless the resting 12-lead electrocardiogram is negative; secondly, if coronary disease is suspected, the single test should be done first; only if this is negative should the double test be performed. If the patient is considered to be acutely ill, it goes without saying that the test should not be done.

We have, we hope, decreased the number of "false" positives by having the tests performed essentially the way a basal metabolism is taken. The patient comes in the morning after a good night's rest; he has no breakfast or, at the most, a light breakfast. He is not permitted to smoke. He is rested a little while on arriving at the office.

In regard to the *ballistocardiogram*, I think the day will come when it will be a more scientific instrument. With physicists and mathematicians working on the machine, trying to improve it, to standardize it, to simplify the technique, I believe that we have a great deal to hope for in the ballistocardiogram. At the present time, however, we must admit that a positive test is observed in hypertension, frequently in apparently normal people over 50, and in people with peripheral vascular disease without heart disease.

There is no specific ballistocardiographic pattern of any type of heart disease although a small "k" has been described in aortic stenosis, coarctation of the aorta and in shock with diminished blood volume in the aorta and its branches. In mitral stenosis, notching of the "k" has been reported. In emphysema the ratio of the inspiratory size of the I-J wave to the expiratory I-J is changed and has been used diagnostically.

An abnormal ballistocardiogram in persons under the age of 50 should be given consideration but it must be correlated with the clinical picture as a whole. Thus, a positive "2-step" exercise electrocardiogram adds weight to the importance of the ballistocardiogram and, in fact, it works both ways.

III. The Vectorcardiographic Diagnosis of Myocardial Infarction

The paper on vectorcardiography has been written by an important investigator in this field. Doctor Wolff gives answers to perplexing questions for which the cardiologist and electrocardiographer have been waiting a long time.

It is true that at present the vectorcardiogram seems complicated and the machines are relatively expensive. In the illustrations the loops of the P-wave, the QRS complex, the terminal junction of the QRS, the RS-T segment and the T-wave all seem jumbled around the point of origin or O point. The vectorcardiogram must be photographed. However, improved equipment is on hand already and the vector curve is now larger and clearer, the components can be separated and prices will become more reasonable. The *"initial,"* and the *"early,"* the *"main"* or *"overall,"* and the *"terminal"* portions of the QRS loop are more easily discerned as sharp entities in the illustrations. There is no doubt that these vector pictures will be still further magnified. Also, the junction or lack of junction of the end of the QRS with the T, that is, the ST segment, will be clearer, thus defining S-T elevations and depressions. A good start on all this has already been made in many laboratories.

Although the "vectorcardiographic" language and the illustrations may be difficult for the general medical reader, he will have gained a good deal if he senses the fundamental principles involved.

The internist and cardiologist are concerned with the future of vectorcardiography. Will it replace the electrocardiogram? Does the vectorcardiogram give a fundamental understanding of the electrical forces in the heart which the electrocardiogram fails to do, or is it good only for teaching, to explain the findings in the electrocardiogram?

There is another important question. Do not the three basic leads, in planes at right angles to each other, called "A, B, C" by Grishman, and "X, Y, Z" by Wolff, give as much information as the vectorcardiogram, particularly, if they are taken simultaneously? May we not employ the VCG to learn more fundamental explanations of the ECG but nevertheless continue to use the ECG regularly, perhaps replacing the 12 leads that we now use by these three fundamental leads? Can we not improve our electrocardiographic interpretation?

This paper marks a landmark for it is the first time that the postmortem findings are correlated with the vectorcardiogram and the electrocardiogram in a considerable series of cases. Doctor Wolff has found the *correlation of the VCG with the postmortem* far superior than in the case of the electrocardiogram. His VCG diagnosis of *septal infarction* was confirmed again and again whereas it had been missed in the electrocardiogram.

Doctor Wolff clearly points out the characteristics of the normal VCG in the three planes and the typical manner in which it is changed in ventricular hypertrophy and bundle branch block and by alterations in the position and rotation of the heart. He then defines the VCG pattern in each type of infarction, distinguishing anterior, high posterior, inferoposterior and lateral wall infarctions, both by the orientation and direction of the abnormal vectors. He shows how the relative positions of the end and the beginning of the vector loop distinguish acute infarction and acute pericarditis and how the shape of the T-wave differs in acute and healing or old infarction.

IV. The Ventricular Pulsations in Myocardial Infarction; A Fluoroscopic and Kymographic Study

For 15 years my colleagues and I have emphasized the ease with which abnormal pulsations of the left ventricle can be observed on fluoroscopy in patients with previous coronary occlusion. We have often pointed out the diagnostic value of abnormal ventricular contractions. However, we have not been successful in "selling" our ideas to the physician. The average practitioner uses the fluoroscope to determine the size, shape and position of the heart but only the exceptional physician has become interested in the pulsations of the left ventricle as evidence of previous coronary occlusion. Perhaps the trouble is that the doctor looks for a large ventricular pulsation whereas in reversal of pulsation at the apex, the movement is probably no more than 1/64th to 1/8th of an inch. At first it requires concentration to see these movements. The patient should take a very short breath and hold it without the slightest strain. For timing, the out-thrust of aorta is considered systole. With a little practice, it is easy to recognize reversal of pulsation due to previous infarction. If there is difficulty, the doctor should move the screen away from the patient, toward himself, thus magnifying the border of the left ventricle. Although he loses light intensity, the magnification more than compensates for this loss.

In addition to reversal of or paradoxical pulsation, the abnormalities to look for are localized diminution or absence of contraction. By "reversal" of pulsation is meant that the border of the left ventricle bulges out during systole, instead of moving in; this finding is almost pathognomonic of an infarct.

Some abnormality in pulsation is found in 75 per cent of cases of previous coronary occlusion. Reversal is found in half the patients. These abnormalities in left ventricular movement are not only of value diagnostically but also prognostically. As the patient improves, we have seen a definite "reversal" become an "absent" or "diminished" and finally a normal pulsation.

The abnormality in pulsation is located in the left lower one-third or one-half of the ventricle. Whether the infarction is diaphragmatic (inferior) or anterior, it is always seen in the usual posterior-anterior position or in a slight left oblique position. Occasionally, the left lateral also demonstrates the abnormality. Reversal of pulsation is found not only in ventricular aneurysm with a true eccentric bulge but also in the ordinary case of coronary occlusion in which the contour is smooth. It is the ordinary case to which reference is made by this reviewer.

We believe that, if the physician develops the habit of using fluoroscopy for this purpose, he will be more than rewarded in that he will have a good means of diagnosing infarction of the left ventricle secondary to coronary artery occlusion. In coronary insufficiency, on the other hand, the infarct is not through-and-through and a reversal of pulsation is not seen.

It has been suggested for many years that the diaphragmatic or inferior border of the left ventricle can be brought into the line of vision by distending the stomach with charged waters. This has proved of help to some.

An interesting future for fluoroscopy lies in improved screen. We have been told that there is a new screen in the making that will give many hundred times the light intensity of the present fluoroscopic screens. When this becomes a fact we will make tremendous advances in fluoroscopy of the heart.

Pulmonary Function in Diseases of the Chest*

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Pulmonary function tests are designed to provide accurate information of the extent and location of disturbed function thereby making available additional information for use with the history, physical, and roentgenological examination in the clinical management of the individual case. A battery of physiological test measurements are used, each possessing a wide range between the normal and the abnormal, and each covering an essential aspect in the evaluation of the adequacy of the gas exchange. One group of tests concerns the bellows action of the chest and lungs and evaluates the ability to move air in and out of the alveoli during the process of breathing. The other group of tests relate to the blood gas exchange across the alveolar-capillary or pulmonary membrane (oxygen and carbon dioxide transport). Pulmonary function measurements assist in the interpretation of the meaning of pulmonary disease from the standpoint of disability, operative risk and treatment.

The efficiency of the bellows action of the chest and lungs for moving air in and out of the alveoli is measured from spirogram tracings and from the residual capacity measurement.[1] Spirogram tracings provide measurements of total vital capacity, timed vital capacity, maximal breathing capacity, and a permanent graphic recording of the exhalation pattern. Evidence of air trapping in the lung is also obtained from the spirogram tracings by having the individual take a deep breath in and then blow out rapidly a few times. Total vital capacity is determined both in the supine and standing position, being the difference between the volume of the lung in the maximal distended position in inspiration and the minimal volume present at the end of a forced maximal exhalation. The total vital capacity in a normal individual is usually slightly larger in the standing than in the supine position. A marked reduction in total vital capacity in the standing position as compared to the supine position indicates the presence of severe pulmonary insufficiency. However, vital capacity when recorded with respect to time becomes a much more significant measurement. The subject is instructed to take in as deep a breath as possible, hold the breath momentarily and then on command blow all of the air out of the lungs as rapidly and as completely as possible. The volume exhaled in the first three seconds measured from the exact beginning of expiration is recorded as the three second timed vital capacity. The three second timed vital capacity is normally the same as the individual's predicted total vital capacity, and represents the maximal functional portion of the vital capacity (a respiratory rate of 15 per minute allows

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four seconds per breath, but if one second is utilized for inspiration only three seconds are left for expiration). The predicted total vital capacity is best computed from the height of the patient.[2]

The maximal breathing capacity reveals the largest volume of air which an individual is able to move in and out of the lungs in a given short unit of time (12-15 seconds), but is expressed as liters per minute. The individual should be instructed to breathe as deeply and as rapidly as possible for 12 seconds, (never longer than 15 seconds), and the rate and depth varied on successive trials (not less than three) to get the maximal value. The 13½ liter respirometer* was developed with the idea of providing an apparatus for mechanical recording with a minimal of breathing resistance for the deep fast breathing and to permit a quick accurate determination of the maximum breathing capacity value (5-10 minutes). The drum on the respirometer has a paper speed which provides a time interval of 12 seconds between the vertical lines. When the rate and depth of breathing are properly varied one can obtain precisely the highest maximal breathing capacity of which an individual is capable and the learning factor as such is not a significant feature. The maximal breathing capacity is a measure of the ability to use the lungs as a bellows and a very wide spread may exist between the normal and the most abnormal cases (10-200 liters per minute). The predicted** maximal breathing capacity is based on the type of apparatus and procedure used and the age, sex, height and weight of the person.[1] If the maximal breathing capacity is normal, the individual is able to use the diaphragm well, the elasticity of the lungs is good, bronchospasm is not a factor and a significant degree of pulmonary emphysema does not exist. The degree of bronchospasm is measured quantitatively by comparing the maximum breathing capacity measurement before and immediately after one bronchodilator treatment using the intermittent positive pressure breathing method for administering a potent bronchodilator substance such as isuprel and vaponefrin.[3, 4] The maximal breathing capacity and the timed vital capacity are independently variable measurements. It has been found in men that if the maximal breathing capacity is above 120 liters per minute, the degree of pulmonary emphysema present is insignificant and significant if less than 40 liters per minute, but between 40 and 120 liters per minute indeterminate.[5] The maximal breathing capacity is a sensitive test and this measurement frequently reveals early abnormalities and hence is valuable for screening purposes, either in the selection of problem cases requiring more complete pulmonary function evaluation or in the detection of poor risks in industrial workers exposed to irritating dusts.[6] The shape of the spirogram tracing as observed on the rapidly moving kymograph drum (32 mm. in 12 seconds) reveals characteristic features of the expiratory flow. A slow and prolonged exhalation curve indicates obstruction to air flow, either of a fixed type or due to bronchospasm and

*Made by Warren E. Collins, Boston, Mass.

**For the 13½ liter Collins respirometer the predicted maximal breathing capacity in liters per minute is men equal (97-Age/2) x BSA in. Sq. meters for women equal (83-Age/2) x BSA in. Sq. meters.

the failure of the end exhalation point of the tracing to return at least to the beginning level indicates air trapping. If the spirogram tracings show a marked rise in the respiratory level during the maximal breathing capacity test with the rapid voluntary respiratory movements, this high inspiratory position indicates the presence of increased breathing resistance and usually the presence of a signficant degree of pulmonary emphysema. Air trapping is a characteristic feature of asthma due to the bronchospasm as the bronchioles are narrower during expiration than inspiration, with a result that expiration is more difficult and prolonged.

The pulmonary function tests described thus far are easy to perform and the only special equipment required is the 13½ liter Collins Respirometer. These tests can be done in the office and on an average only five to 10 minutes are required to obtain satisfactory spirogram tracings. When the degree of bronchospasm is evaluated a slightly longer time period is required, since the bronchodilator should be administered over a period of at least 10 minutes to be certain of obtaining the maximal results.[3, 4] These tests are useful in evaluating the response to treatment, in following the clinical course of a disease by comparing serial spirogram tracings taken at intervals (similar to the chest roentgenogram) and in screening tests of candidates for surgery, anesthetic risk, workers exposed to lung irritants (either dust or gases) and other problem types of cases.

A second important basic measurement relating to the bellows action of the chest and lungs is the residual air capacity, which by definition is the volume of air remaining in the lung at the end of a maximal forced exhalation. The residual air occupies normally about 25 per cent of the total lung capacity, being slightly smaller in the younger age group (20 up to 35 years) and slightly larger in the older age group (30 over 60 years). In pulmonary emphysema the volume of residual air is increased in relation to the total lung volume and if this ratio is over 35 per cent a significant degree of pulmonary emphysema exists. The residual air is measured quantitatively using the oxygen open circuit method, and checked routinely on two runs to within 100 ml., if not the run is repeated. If the alveolar air contains more than 1.5 per cent nitrogen after breathing oxygen for seven minutes, impairment in the uniformity of air distribution in the lung exists.[1] The ratio of the residual air to the total lung capacity is most important in the individual case since this corrects for changes in total lung volume which may exist (either normal, increased or decreased). If the residual air occupies 35-45 per cent of total lung capacity a moderate degree of emphysema is present, from 45-55 of total lung capacity an advanced or severe degree and above 55 a far advanced or very severe degree of emphysema exists. The accurate diagnosis of pulmonary emphysema in some cases requires measurements of residual air, as the physical examination and the chest roentgenogram may be misleading or inconclusive. Pulmonary emphysema is frequently missed on the chest roentgenogram when a severe degree actually exists.

The three measurements of pulmonary function thus far described of greatest importance relating to the bellows action of the chest and lungs

and measuring independently variable aspects are: (1) the timed vital capacity for three seconds, (2) the maximum breathing capacity and (3) the residual per cent of total lung capacity.

A numerical ventilation factor (V.F.) has been used to evaluate the efficiency of the chest and lungs as a bellows from the average of the three measurements, all expressed as per cent of the normal predicted, namely: the three second vital capacity, the maximal breathing capacity and the residual air as per cent of total lung capacity. The ventilation factor provides a single figure value of the patient's ability to use the chest and lungs as a bellows for aerating the alveoli and this is well correlated with the arterial pCO_2 in mm. Hg. as determined by direct tension measurements.[6]

The second basic aspect of pulmonary function, namely the blood gas exchange across the alveoli-capillary membrane, is evaluated from studies of the arterial blood and the expired air. Normally, at sea level pressure, the arterial blood hemoglobin is 96-98 per cent saturated with oxygen both at rest and during exercise. The arterial blood oxygen saturation is obtained by dividing the corrected oxygen content by the corrected oxygen capacity as volumes per cent for each sample of blood. The Van Slyke manometric apparatus is used for the measurements of oxygen content and capacity. The double scale oximeter* using the cuvette appears to be a fairly satisfactory procedure for determining the arterial blood oxygen saturation. A drop in the arterial blood oxygen saturation of 5 to 10 per cent or more below the resisting level with exercise (after the one minute step-up test) indicates severe disability.[1] An increase in the exercise oxygen saturation above the resting level indicates less disability than implied by the resting level measurement.[7] It is thus of great importance to compare in each case where possible the rest and exercise arterial blood oxygen saturation, as often the exercise arterial blood oxygen saturation is of much greater value than the resting in evaluating the pulmonary function status. If the exercise arterial blood oxygen saturation drops 5 to 10 per cent below the resting level the individual should be restricted in such activities as walking up steps or up grades. In general, the exercise saturation tends to decrease in severe emphysema, but this is not always the case, and in pulmonary fibrosis without emphysema, a marked drop in the exercise arterial blood oxygen saturation frequently occurs.

An elevated arterial CO_2 content indicates difficulty in blowing off CO_2 and warns of the possibility of developing respiratory acidosis during infections or if the respiration be depressed by sedative drugs, anesthetics, or by other means. The arterial blood pH is the best single measurement revealing the exact status of the acid base balance concerning the presence of acidosis or alkalosis. If the patient has a marked degree of arterial blood oxygen unsaturation with an elevated CO_2 content, then the administration of high oxygen concentrations with a mask or even with a catheter or tent may precipitate respiratory acidosis, drowsiness and even

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coma or death.[8] In such a patient the sudden change to high oxygen breathing decreases the minute ventilation as a result of removing part of the stimulus to the respiratory center from the carotid bodies, as these are stimulated by the low oxygen in the blood and reflexively stimulate the respiratory center. The high oxygen breathing relieves the arterial blood unsaturation and removes the stimulus from the carotid bodies and the ventilation decreases, in some to a critical level unless supplemented by mechanical aids.

The expired air from the patient is studied with respect to the minute ventilation (total volume of air breathed during a test period of three to five minutes), the total oxygen uptake and the per cent of oxygen extracted from the inspired air breathed. The minute ventilation is measured with respect to the number of liters per minute per square meter of body surface area. If the minute ventilation is below the normal range, hypoventilation is indicated and if above hyperventilation. In some cases hyperventilation may be the only abnormality revealed from the pulmonary function evaluation. The one minute step-up exercise test (30 steps on an eight inch stool in one minute) has been standardized with respect to the normal range. In some cases of marked pulmonary insufficiency the exercise minute ventilation may be inadequate in proportion to the step-up test requirement. In general, the minute ventilation as a single measurement is often indeterminate as revealed from extensive statistical studies on a large series of cases. The minute ventilation along with the data provided from the expired air gas analysis provides the necessary data to calculate the oxygen uptake. The oxygen uptake is determined both at rest and with exercise. The resting oxygen uptake tends to be in the normal range regardless of the degree of pulmonary insufficiency, and hence, is of little value in pulmonary disability evaluation. On the other hand, the measurement of step-up exercise oxygen uptake is a very significant value. A marked reduction in the exercise oxygen uptake in the presence of an adequate minute ventilation reflects the inability of the individual to expand the pulmonary bed and increase the pulmonary blood flow or to increase the cardiac output from the right heart to the elevated level representing a normal response proportionate to the degree of exercise given.[1] If the normal amount of oxygen is removed from the expired air during the one minute step-up exercise an increased blood flow is necessary. If the oxygen uptake is normal during exercise this indicates that the pulmonary blood flow is increased in proportion to the degree of exercise and that the pulmonary vascular resistance is not significantly altered. A decreased oxygen uptake during step-up exercise indirectly indicates increased pulmonary vascular resistance, without the necessity of catheterizing the right heart to measure mean pulmonary arterial pressure and the cardiac output.

The per cent of oxygen extracted from the inspired air breathed is a measure of the lung ventilation efficiency, and this figure is used to determine the oxygen uptake along with the pulmonary ventilation volume measurement. Normally 4 to 5 per cent of the oxygen is extracted from

the inspired air at rest and 5 to 6 per cent during the step-up exercise. Thus, if a patient is removing only 2.5 per cent of the oxygen from the inspired air during exercise, twice the normal volume of air would be required for breathing to furnish the oxygen. In chronic pulmonary disease the per cent of oxygen extracted from the inspired air is usually decreased somewhat, but this measurement is usually indeterminate as to the degree of pulmonary function impairment present unless the decrease is of marked extent. Excitement with hyperventilation may result in a low abnormal value, which might be normal if the individual were breathing at a lower volume rate. A severe degree of pulmonary insufficiency may exist with hypoventilation present during step-up exercise and a normal per cent of oxygen extracted, but the total oxygen uptake would be decreased markedly.

The duration of dyspnea with a standard exercise test (such as the one minute step-up) is of value in pulmonary function evaluation. The one minute step-up test increases the oxygen uptake normally three to four times above the resting level and is designed as a test of pulmonary function rather than physical fitness. This test is satisfactory for routine use in studying patients with chronic pulmonary disease. The exercise study is of great value in demonstrating disturbances in ventilation-perfusion relationships (changes in the arterial blood oxygen saturation and total oxygen uptake). The duration of the dyspnea with the one minute step-up test is a subjective sensation on the part of the patient and it is subject to limitations as such, but careful observations by trained personnel minimize the subjective element. For the normal individual, the one minute step-up test is a mild exertion and the subjective dyspnea is less than 90 seconds in duration. A marked prolongation of the dyspnea constitutes a significant finding and indicates a severe degree of function impairment either cardiac or pulmonary. In general, the pulse and the respiratory rates are unreliable measurements on which to assess pulmonary function impairment. Statistical analysis of a large series of data on respiratory rate, pulse rate, and minute ventilation has revealed the inadequacy of these measurements alone as shown by the magnitude of the standard deviation.[9] Breath holding time has been found to be of no value. Tests of circulation times with ether and decholin, blood volume, hematocrit, hemoglobin level and venous pressure, and the electrocardiogram are frequently very helpful in differentiating the pulmonary and cardiac aspects of a given case.

Great advances have been made in research investigations relating to pulmonary function in recent years and many of these new tests are interesting and give promise of clinical value in the future, but as yet they are not sufficiently well established or the necessity proved to be routinely included in a pulmonary function evaluation. The direct tension measurements[10] of arterial pO_2 and pCO_2 is a valuable research procedure, but it is difficult to acquire a satisfactory proficiency in performance, hence subject to many errors by inadequately trained personnel. The use of the nitrogen meter[11] appears promising, but as yet the practicability and reli-

ability of both test and apparatus have not been adequately demonstrated. Pulmonary compliance,[12] relating to the pressure volume relationship of the lung, is a popular study at the present time, but whether this will provide any more practical clinical information over that which can be obtained with simple spirogram tracings remains to be demonstrated. Similarly the pneumotachograph is an interesting research instrument, but whether it provides any more information than can be obtained from the spirogram tracings on the respirometer remains to be demonstrated.

SUMMARY

An accurate evaluation of the degree of pulmonary function impairment may be made from the following physiological tests: (1) Ventilation measurements from spirogram tracings (total vital capacity, three second timed vital capacity, maximal breathing capacity and the shape of the exhalation curve following a deep breath), (2) the degree of bronchospasm present, (3) the residual air capacity and alveolar nitrogen per cent after seven minutes oxygen breathing, (4) the arterial blood oxygen saturation at rest and immediately after step-up exercise, (5) the oxygen uptake during step-up exercise, (6) the per cent of oxygen extracted from the inspired air breathed and (7) the character and duration of dyspnea after step-up exercise.

RESUMEN

Una exacta valuación del grado de daño a la función pulmonar, puede lograrse mediante las siguientes pruebas funcionales: (1) Medidas de ventilación por los trazos espirométricos (capacidad vital total, capacidad vital en tiempo de tres segundos, capacidad vital máxima y la forma de la curva de la exhalación después de una respiración profunda), (2) el grado de broncoespasmo presente, (3) la capacidad de aire residual y el nitrógeno alveolar por ciento después de siete minutos de respirar oxígeno, (4) la saturación de oxígeno de la sangre en reposo e inmediatamente del ejercicio del escalón, (5) la toma de oxígeno durante el ejercicio del escalón, (6) el porciento de oxígeno extraído del aire respirado y (7) el carácter y la duración de la disnea después del ejercicio del escalón.

RESUME

Les troubles de la fonction pulmonaire peuvant être évalués par les tests physiologiques suivants: (1) mesure de la ventilation par tracés siprographiques (capacité vitale totale, capacité vitale mesurée pendant trois secondes, capacité ventilatoire maxima et aspect de la courbe expiratoire suivant uné respiration profonde), (2) degré du bronchospasme existant, (3) capacité de l'air résidual, et du pourcentage d'azote alvéolaire après sept minutes de respiration dans l'oxygène, (4) saturation oxygénée du sang artériel au repos, et immédiatement après des exercices de montée d'escalier, (5) augmentation de l'absorption d'oxygène au cours des exercices de montée d'escalier, (6) pourcentage d'oxygène retiré de l'air inspiré et (7) caractère et la durée de la dyspnée après exercice de montée d'escalier.

REFERENCES

1 Motley, Hurley L.: "The Use of Pulmonary Function Tests for Disability Appraisal: Including Evaluation Standards in Chronic Pulmonary Disease," *Dis. Chest*, 24:378, 1953.

2 Baldwin, E. deF., Cournand, A. and Richards, D. W. Jr.: "Pulmonary Insufficiency. I. Physiological Classification, Clinical Methods of Analysis, Standard Values in Normal Subjects," *Medicine*, 27:243, 1948.

3 Motley, Hurley L., Lang, Leonard P. and Gordon, Burgess: "Use of Intermittent Positive Pressure Breathing Combined with Nebulization in Pulmonary Disease," *Am. J. Med.*, 5:853, 1948.

4 Motley, Hurley L. and Tomashefski, Joseph F.: "Treatment of Chronic Pulmonary Disease with Intermittent Positive Pressure Breathing," *Arch. Indust. Hyg. and Occupational Med.*, 5:1, 1952.

5 Motley, Hurley L. and Tomashefski, Joseph F.: "Studies on the Maximal Breathing Capacity Measurement," *Am. J. Physiol.*, 163:736, 1950.

6 Motley, Hurley L.: "Clinical Pulmonary Physiology II. Detection of Early Lung Function Change in Industrial Exposure," *Idust. Med and Surg.*, 22:6, 262, 1953.

7 Motley, Hurley L. and Tomashefski, Joseph F.: "Comparison of Treadmill and Step-up Exercise Using Arterial Blood and Respiratory Gas Exchange Measurements," *Federation Proceedings*, 9:92, 1950.

8 Motley, Hurley L.: "Oxygen Therapy," *Veterans Administration Technical Bulletin, TB 10-95 Dec.* 31, 1953.

9 Motley, Hurley L.: "Evaluation of Pulmonary Function-Impairment by Physiological Studies in 400 Unselected Cases of Hard and Soft Coal Miners with Respiratory Complaints. Conference on Silicosis and Aluminum Therapy, Schumaker, Ontario, Canada, Jan. 30, 1951." *Toronto, McIntyre Research Foundation* 1951, pp. 49-67.

10 Riley, R. L., Proemmel, D. D. and Franke, R. E.: "A Direct Method for Determination of Oxygen and Carbon Dioxide Tensions in Blood," *J. Biol. Chem.*, 161:121, 1945.

11 Fowler, Ward S.: "Lung Function Studies, III. Uneven Pulmonary Ventilation in Normal Subjects and in Patients with Pulmonary Disease," *J. Applied Physiology*, 2:283, 1949.

12 Lindgren, Inga, Mead, Jere, Gaensler, Edward A. and Whittenberger, James L.: "Pulmonary Mechanics in Emphysema," *Clinical Research Proceedings*, 1:115, 1953.

The Time Factor in Anesthesia*

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Not more than 100 years ago, that is, before the discovery of anesthesia, time was the essence in doing surgery. Twenty minutes was the longest time any type of surgery could possibly be endured by a patient. After that time, the patient was likely to die from exhaustion or shock. "Have everything ready and do not lose time"—was the motto of the surgeon. Even 10 and eight years ago, it had great advantage for a patient to be operated by a surgeon who could work fast. At that time, only deep, third plane anesthesia could supply necessary relaxation, and we know that one-half hour of third plane anesthesia can mean shock for a patient who can easily endure four to five hours of light anesthesia. Ether, the anesthetic agent most commonly used to procure relaxation, has a considerable depressant effect on the myocardium in deep anesthesia; none in light anesthesia. The same is true for other agents. Moreover, during deep anesthesia, all the available anesthetic agents produce a profound depression of peripheral homesostatic mechanisms.

Recently, Rovenstine and his associates[1] demonstrated in dogs by direct microscopic observations of a portion of exteriorized omentum that, under light anesthesia by three different agents (cyclopropane, ether, sodium pentothal), the capillary circulation remained relatively unaffected; vasomotion of the metarterioles and precapillaries was preserved and venous return was rapid. The caliber of the small arterioles was normal with no appreciable dilatation, indicating good vasoconstriction. When a deep phase of anesthesia was reached, considerable impairment of the capillary circulation was seen while vasomotion was rapidly depressed and disappeared within one or two minutes. The response of the precapillary sphincters to epinephrine was depressed; circulation through the capillary bed was plethoric throughout; blood flow in the collecting venules was seriously slowed; and marked arteriolar dilatation was noted. Thus, the deteriorating effect of deep anesthesia was clearly demonstrated.

Fortunately, deep planes of anesthesia are not needed today. We can carry the patient in light anesthesia and supply the necessary relaxation by muscle-relaxant drugs. It could be shown in experimental and clinical work, and it is our own experience, that there is no deterioration of the homeostatic mechanism and no depression of the myocardium when muscle relaxants are used in adequate, non-excessive doses, if oxygenation and tidal exchange of the patient are taken care of adequately, and blood loss is replaced as it occurs.

Thus we can maintain a patient in good condition for any length of time in light anesthesia. Today there is no need for the surgeon to hasten.

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He can be gentle and take whatever time is needed to perform his task, however delicate. There is no reason for the surgeon to rush except in order to stem unforeseen and excessive bleeding, and there should be no reason for the anesthesiologist to rush, except if something is wrong with the patient. Hypoxia should not be tolerated; obstruction should be diagnosed and taken care of immediately; deleterious positions should be corrected at once. On the other hand, the qualifications of an anesthesiologist should not be judged by how fast he is ready for the surgeon.

The statement has been made not long ago that the great success in a series of heart operations was mainly due to fast intubation. Even more amazing is the fact that a group of good anesthesiologists preface their original article with the following statement[2]:

> "The adoption of the routine use of detergents for the surgical scrub has deprived the anesthesiologist of the minimal 10 minute period for induction and intubation which was previously available to him. At present the interval of time which elapses from the time the surgeon sees his patient immediately preoperatively, until he is ready to begin the operation averages five minutes."

Thus these anesthesiologists draw the conclusion that these five minutes have to suffice for them to get the patient ready for the operation. Fifteen to 20 minutes may be necessary to put the patient in the correct position (say in chest surgery); the operation may last four to five hours, but five minutes is all the anesthesiologist gets to be ready. For this reason, these authors recommend their method of fast intubation. They use rapid injection of a predetermined amount of an ultra-short acting barbiturate combined with a curare preparation, a standard mixture of 400 mgr. thiopentothal (8 cc. of a 5 per cent solution) and 4 mg. of deca-methonium bromide. The 5 per cent solution of sodium pentothal instead of the usual 2½ per cent is chosen to increase the rapidity of the injection, the entire injection thus requiring five seconds. After 20 to 25 seconds the patient can be intubated. The time required for induction and intubation using this technique was not permitted to exceed 60 seconds. Many similar procedures are being recommended.

One may question whether the fact that the surgeon is ready after five minutes of scrubbing is a reason to hasten induction and intubation to such a degree, and whether there are any advantages to the procedure. How does this method compare with the more conservative procedures, where induction is accomplished by intermittent injection of an ultra-short acting barbiturate watching the patient's blood pressure closely, and stopping when the lidreflex disappears; keeping the patient breathing and well oxygenated for several minutes while supplying small amounts of ether or gas; and eventually injecting the selected type of curare before intubation, taking five to 10 minutes, instead of 25 seconds.

In a paper evaluating a new ultra-short acting barbiturate (surital sodium) Phillips[3] states that, immediately following the injection of the drug, a drop in blood pressure is noted proportional to the rapidity of the injection and to the larger initial dose. Stephen et al,[4] in an article on

"Hypotension under Anesthesia" state that rapid injection of ultra-short acting drugs into the bloodstream may produce specific effects in the cardiovascular system of man, including a drop of blood pressure and a reduction of cardiac output due to direct depression of the myocardium. A considerable fall of blood pressure was noted by the writer[5] after rapid intravenous injection of sodium seconal; this is not present when the drug is injected slowly.

In order to clarify the problem, the Duke group of Anesthesiologists[6] set up an excellent investigation. Two hundred and thirty-eight patients were given the "shot gun" method under carefully controlled conditions. Twenty-two had the conventional slower induction. E.C.G., blood pressure, and pulse rates were recorded preoperatively, after injection of the muscle relaxant, and again after injection of the barbiturate. There was a persistent fall in blood pressure, whether sodium pentothal, sodium surital, or evipal was administered rapidly; a decrease of 40 mm. Hg. or more of the systolic pressure took place in about 1/3 of the patients. In five patients, blood pressure could not be obtained in the interval between administration of the hypnotic and laryngoscopy. One good-risk patient developed cardiac arrest two minutes after the barbiturate was injected, and in two patients of this series, operation had to be cancelled due to persistent hypotension. It is interesting that a 40 to 50 mm. fall in blood pressure was also noted in the series of the group that advise the "shot gun" method of intubation in order to save time.

On the other hand, not one of the 22 patients observed by the Duke group using the intermittent administration of sodium pentothal, followed by the intravenous use of a muscle relaxant and nitrous oxide inhalation, had a fall in blood pressure of more than 20 mm. Hg., a fact which confirms our observations. There were no significant changes in the E.C.G.s of these 28 patients.

In both series of patients investigated during rapid injection of the barbiturates, the blood pressure fall was rectified, with only four exceptions, during intubation. The blood pressure rapidly rose to preanesthetic levels, together with an increase in pulse rate, probably due to sympathetic impulses caused by laryngoscopy and intubation. The latter were accompanied by arrhythmias. It is well known that direct laryngoscopy and tracheal intubation uncomplicated by coughing, anoxia or hypercapnia, may produce considerable rise in blood pressure and an increase in heart rate. Dripps and his co-workers[7] state that this rise in blood pressure and the cardiac acceleration represent a stress placed upon the myocardium. They could record rises in systolic pressure of more than 90 mm. Hg. during intubation under light anesthesia, and therefore advise intubation in second or third plane anesthesia.

The considerable fall in blood pressure followed by such a considerable rise may often escape the attention of the anesthesiologist using the rapid intubation method. After all, only one or two minutes are involved, he is very busy, and when he takes the blood pressure after intubation, the preanesthetic level has been reached, and the patient seems in good condi-

tion. Two wrongs seem to make one right, and apparently in most cases one can get by with it. This is certainly not true in all cases: obviously not in a patient with a severe mitral stenosis, who has a relatively fixed cardiac output; or in a patient with advanced aortic stenosis where adequate circulatory compensation cannot be maintained after such rapid changes. Likewise, a heart damaged by previous myocardial infarction poorly tolerates rapid alterations of circulatory dynamics.[2] Even in a normal heart the rapid alterations in cardiac function following rapid injection of a potent ultra-short acting barbiturate cannot be ignored. Moreover, it cannot be predicted which patients would react with the greatest fall of blood pressure, and which would have more numerous irregularities.[6] There seems to be no direct correlation as to age or general status of the patient. Besides, and most astonishing, no relationship can be demonstrated between the appearance or not of the undesired effects, and a smooth intubation or a difficult one with bucking or coughing on the tube or even laryngospasm.

For these reasons, it is more desirable to administer the ultra-short acting barbiturate intermittently over a longer period of time, and to attempt laryngoscopy and intubation in a slightly deeper plane of anesthesia, by introducing potent analgesic drugs like ether or cyclopropane for a short time. Thus, it is desirable to take a few more minutes to get ready for the surgeon. The safety of the patient should be the first consideration of the surgeon *and* of the anesthesiologist. As the time factor fortunately no longer plays an important role in considering the alloted time for a surgical procedure, it should not be given any importance in itself in getting the patient ready for surgery.

SUMMARY

Light anesthesia with the common anesthetic agents does not significantly interfere with the homeostatic mechanisms, nor does it depress the myocardium. The same is true for adequate amounts of muscle relaxant agents. Problems of respiratory physiology and blood replacement are well understood. Thus, practically limitless operating time can be afforded the surgeon. Therefore, no undue pressure should be exerted on the anesthesiologist limiting the time allotted for induction and intubation.

Various dangers inherent in the method of rapid intubation are pointed out and discussed.

Intermittent intravenous injection of the barbiturate and addition of small amounts of potent inhalant anesthetic agents for intubation in a slightly deeper plane of anesthesia are recommended. This would avoid those rapid changes of circulatory dynamics which are encountered when using the "gun shot" method of induction and intubation.

RESUMEN

La anestesia ligera con los agentes comunes, no interfiere de modo importante con los mecanismos homeostáticos ni deprimen el miocardio. Lo mismo ocurre con respecto de los agentes relajantes del músculo a dosis

adecuadas. Así, práticamente el cirujano dispone de tiempo ilimitados. Por tanto, no debe hacerse presión sobre el anestesista limitado el tiempo concedido para la inducción y la intubación.

Se señalan varios peligros inherentes al método de la intubación rápida y se discuten.

La inyección intravenosa de barbitúricos y el agregado de pequeñas cantidades de agentes anestésicos potentes por inhalación para la intubación en un plano ligeramente más profundo de anestesia, se recomiendan. Esto evitaría los rápidos cambios de la dinámica circulatoria que se encuentran al suar al método repentino de inducción e intubación.

RESUME

Une anesthésie légère avec les produits habituels n'a pas d'action importante sur le mécanisme circulatoire et ne cause pas de troubles myocardiques. Les mêmes constatations s'appliquent lorsqu'on utilise à dose correcte l'action des agents susceptibles de créer la relaxation musculaire. Les problèmes de la physiologie respiratoire, et de la transfusion de remplacement sont bien établis. Ainsi le chirurgien peut se permettre d'opérer pendant un intervalle de temps pratiquement illimité. C'est pourquoi on ne devrait pas insister auprès de l'anesthésiste pour limiter le temps nécessaire à la mise en train de l'anesthésie et à l'intubation.

L'auteur met en évidence les différents dangers qui peuvent survenir lorsque l'intubation est faite rapidement, et il en discute la signification.

L'auteur préconise des injections intra-veineuses intermittentes de barbiturique et l'adition de petites quantités d'agents anesthésiques par inhalation pour l'intubation de façon à réaliser un plan d'anesthésie très progressivement croissant. Ainsi seraient évitées les modifications rapides de la dynamique circulatoire qui surviennent lorsqu'on pratique une méthode de mise en train de l'anesthésie et d'intubation brutale, en "boulet de canon."

REFERENCES

1 Hershey, S. G., Zweifach, B. W. and Rovenstein, E. A.: "Effects of Depth of Anesthesia on Behavior of Peripheral Vascular Bed," *Anesthesiology*, 14:245, 1953.

2 Colon-Yordon, E., MacKrell, T. N. and Stone, H. H.: "An Evaluation of the Use of Thiopentothal and Decamethonium Bromide for Rapid Endotracheal Intubation," *Anesthesiology*, 14:255, 1953.

3 Phillips, H. S.: "Some Clinical Observations on the Use of Surital Sodium in Combination Anesthesia," *Current Researches in Anesthesia and Analgesia*, 32:56, 1953.

4 Stephen, R., Nowill, W. K. and Martin, R.: "Diagnosis and Treatment of Hypotension During Anesthesia," *Anesthesiology*, 14:180, 1953.

5 Weyl, R., Liu, Wei-chi and Lipp, E.: "Evaluation of the Usefulness of Intravenous Sodium Seconal in Anesthesia," *Journal of International College of Surgeons*, 18: 365, 1952.

6 Stephen, C. R., Martin, R., Nowill, W. K.: "Cardiovascular Reactions of Surital, Pentothal or Evipal Combined with Muscle Relaxants for Rapid Anesthesia Induction," *Current Researches in Anesthesia and Analgesia*, 32:361, 1953.

7 King, B. D., Harris, Jr., C. L., Greifenstein, F. E., Elder, Jr., J. D. and Dripps, R. D.: "Reflex Circulatory Responses to Direct Laryngoscopy and Tracheal Intubation Performed During General Anesthesia," *Anesthesiology*, 12:556, 1951.

Palliative Procedures for the Treatment of Carcinoma of the Esophagus*

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Not too many years ago, it was predicted that the esophagus would never yield to surgical attack. Development of improved technics in thoracic surgery and related specialties has made possible the resection of the esophagus with decreasing rates of mortality and morbidity. A state of optimism then arose in association with treatment of cancer of the esophagus. Surgeons have been developing more extensive procedures for managing the more advanced forms of cancer. Longer lengths of the esophagus can be resected successfully now. The structures within the mediastinum are involved by carcinoma early; although the esophagus may be resected, the related structures cannot be.

Surgical technics now make possible the resection of any portion of the esophagus and the re-establishment of continuity at any level in the thorax or cervical region by anastomosing either the mobilized stomach, jejunum, or colon to the proximal esophageal segment. The reconstructed esophagus may be placed in either the old esophageal bed, the right or left pleural cavity, the anterior mediastinum, or in the subcutaneous tissue of the chest.

Results in the treatment of carcinoma of the esophagus by surgery and by other means have been discouraging. However, there have been enough cures or long-term survivals to give hope to the patient and doctor. When the esophageal growth appears resectable and the patient's physical condition adequate, the patient should be offered surgical therapy. This does not mean though that the surgeon is to make an heroic effort to resect a non-resectable lesion or to do a more extensive procedure than the physical status of the patient can tolerate or the end result can justify.

In a few patients at the time of the clinical work-up, and in many patients at the time of exploration, it is discovered that the lesion in its entirety cannot be resected. For this clinical group many surgeons yet will advocate resective surgery with removal of as much of the tumor as possible. For a large percentage of these cases, there are indications that the same results can be obtained with procedures of lesser surgical magnitude. Also, there are many patients who, because of unrelated diseases in other organs, are unable to tolerate major degrees of surgical trauma.

In any case, the surgeon is confronted with the responsibility of re-establishing or maintaining the oral intake of food and water. There are today palliative technics of varying degrees of surgical magnitude

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for restoring or preserving the patency of the esophagus. There is little excuse for refusing a patient one of these latter procedures which have a comparatively low incidence of mortality and morbidity.

These procedures can be used as a final therapeutic agent or as a preliminary stage of a more radical approach:

1. *Gastrostomy or Jejunostomy:*

Used as a palliative measure in a patient with a non-resectable esophageal lesion, gastrostomy or jejunostomy prevents starvation only. There is a relatively high postoperative mortality associated with these procedures. Obstruction is not relieved; the source of pain is not removed, nor is prevented the accumulation of the troublesome pharyngoesophageal secretions which continue to be aspirated into the tracheobronchial tree, preventing comfort or sleep. Gastrostomy feedings have a demoralizing effect upon the patient. There is an indication for a gastrostomy or jejunostomy only in the patient who cannot tolerate a more extensive surgical procedure and at the same time has such a severe curvature of the spine that endoscopy cannot be carried out.

As a pre-resective measure, gastrostomy or jejunostomy cannot be considered effective procedures for getting a patient in better nutritional status.

In conjunction with retrograde dilatations with Tucker bougies during and following deep therapy, there may be an indication for a Spivack gastrostomy. In post-irradiation patients, it is possible that the lumen could be maintained by this method in a large percentage of the cases of the more stenosing, fibrous lesions.

II. *Endoscopy and Dilatations:*

Esophagoscopy and gentle, careful dilation of the obstructed lesions with one of the various types of dilators can produce immediate relief. This procedure has less associated risk than a resection, particularly a high resection in an emaciated, dehydrated patient. Vinson reports a perforation rate of only 1 per cent.

It seems unfortunate that many thoracic surgeons, for various reasons, themselves do not perform esophagoscopies or dilate carcinomatous lesions. It would seem that the same surgeon, at the time of esophagoscopy or at the time of thoracotomy, weighing the indications for the procedures and the risks of mortality, would rather accurately evaluate the situation into which he would be placing himself.

Dilatations should be used more often for the purpose of getting a patient with an obstruction in good physical condition for surgery. If a repeat esophagoscopy is necessary, a catheter may be passed into the stomach for feeding purposes. Gastrostomies can be avoided except in rare cases.

There is an indication for dilatation also in those patients who are considered candidates for irradiation therapy. Adequate roentgen therapy in poor risk patients has an associated high mortality. The use of a catheter for a few days following dilatation will prevent re-obstruction

by the tumor or by temporary congestion and edema which could be expected to take place at the site of irradiation. In the case of partial obstruction, a thread can be left in the lumen at the time of dilatation to act as a guide for subsequent dilatations should deep therapy produce post-irradiation swelling.

Esophagoscopy with dilatation often can maintain an opening in a post-irradiation case. I have used the Hurst mercury bougie for this in some cases, and, in others, the esophagoscope with Jackson dilators. The method of anchoring a silk thread in the intestine and passing threaded dilators along this guide has been used by Vinson. Dilatation therapy for lesions immediately adjacent to the left bronchus or the aortic arch should not be used, because of the possibility of producing bronchial or aortic fistula. Palmer cautions against using a larger dilator than the number 18 French bougie, while Vinson has used a number 40 French bougie.

III. *Intubation:*

Carter Symonds was the first to intubate the esophagus, using a gum-elastic tube. In 1927, Souttar reported an improved tube for this purpose which consists of a close spiral of German silver wire. The proximal end of this tube has a collar which tends to prevent its onward passage. Its flexibility enables it to conform to the lumen of the esophagus. Allison and Borrie, who re-evaluated this method in 1949, considered the Souttar tube still of usefulness in selected cases. In 1949, Brown reported on the use of a silver tube which he had inserted orally into the esophagus. Ravitch has reported a case in which, following an exploration of a non-resectable lesion, a plastic tube was inserted orally through the stricture before the chest was closed. In selected cases intubation can afford many months of relief for the patient before distant metastasis or complications prove fatal.

IV. *Local Excision of the Obstructive Growth and Bridging of the Defect with a Prosthesis:*

In 1922, Neuhof and Ziegler performed in experimental animals a two-stage procedure which consisted, in the first step, of packing off a local area of the esophagus to produce a granulation tissue barrier. At a second stage, the esophagus was resected and the defect bridged with a rubber tube. Berman, since that time, has modified this work by performing a local excision of the esophagus and immediately bridging the defect by using a flanged, flexible polyethylene tube. This technic has been used in a small group of patients with favorable results.

It would seem that indications for this procedure could be found in the following instances: at the time of exploration, when the lesion is found to be non-resectable; in the case of the patient with low surgical reserve; and, during surgery, should situations arise that make it unlikely that the patient can withstand a continuation of extensive resection.

V. *Shunting Technics:*

Non-resectable lesions of the lower esophagus can be by-passed readily by severing the esophagus above the lesion and carefully closing the distal

end. The proximal end can then be anastomosed, less often to a loop of the jejunum, and more often to a Roux-Y segment, which can be brought up through the diaphragm and lateral to the esophageal opening. Nonresectable lesions at higher levels can be treated similarly by primary anastomosis with a longer Roux-Y segment. An alternative is Yudin's technic of bringing the jejunum through a subcutaneous tunnel to the cervical esophagus and performing the anastomosis at a second stage.

VI. *Irradiation Therapy:*

Irradiation therapy can be given by radium bougie, direct implantation of radioactive material, or deep irradiation using various voltages and technics. Discouraging surgical results, particularly in the experience with the high thoracic and cervical lesions, are forcing a re-evaluation of irradiation therapy. There are two facts concerning irradiation that are worth remembering: 1) Following therapy many patients rapidly recover ability to swallow. This recovery often remains for the duration of the illness, although death occurs from extension of the tumor. 2) Following therapy death frequently occurs from perforation of the esophagus either into the mediastinum, pleural cavity or aorta. Autopsy in such cases has revealed no evidence of residual carcinoma. These facts suggest certain therapeutic possibilities:

Used as a preoperative measure, short periods of deep therapy will relieve obstruction and clear up much of the sloughing, ulcerating and infected tumor. Having had a period in which there could be normal oral intake of food and fluids, a patient could come to surgery in good nutritional and mental balance. I have had two patients who received preoperative irradiation therapy. Both underwent palliative intrathoracic esophagogastrostomy. There is no question that their preoperative condition was greatly improved by irradiation, and I do not believe that the dissection was more difficult because of it.

The idea of resection and postoperative deep therapy has been suggested by others, and a controlled series should be evaluated.

As a curative or palliative method, several technics for administering irradiation have been suggested:

In Denmark, where little esophageal surgery is done, Neilson advocates the technic by which the patient is placed on a special stool and rotated with the esophagus as the axis of rotation. Rotation takes place at right angles to the field of irradiation, in order that the maximum dosage is delivered to the tumor with a minimal dosage to the skin. Twenty-five per cent of his cases were alive at the end of one year, and 15 per cent, at the end of two years. Sweet's surgical series, which may be more selective, reveals 42 per cent alive at one year, and 34 per cent, at two years.

In this country the multiple-port method for irradiating esophageal lesions has produced comparative results.

Negus has used a local irradiation technic. Using the esophagoscope, he gently dilates the lesion with a bougie and measures the exact length of the tumor mass. Radium or radon seeds are secured at one-centimeter intervals along a small rubber or plastic tube that projects one centimeter

beyond either end of the growth. Three to four of these tubes are inserted and left for a period of approximately 10 days. It would be interesting to see a series of cases treated by this method in conjunction with external irradiation.

SUMMARY

The importance of careful selection of the therapeutic plan for a given case of esophageal carcinoma is emphasized. There must be on the part of the physician and surgeon a willingness and ability to modify a given plan should unfavorable situations exist or arise. There will continue to be many cases in which major curative or palliative resections cannot be done, either because of the general physical condition of the patient or because of the non-resectability of the esophageal growth. In these cases, other procedures of lesser magnitude than a resection can and should be used.

Longer survival and more comfort to a greater number of patients can result from making proper choices of the curative and palliative technics now available for treatment of carcinoma of the esophagus. The patient who is discharged from the hospital with a gastrostomy, who either has been refused esophageal therapy or has been given an exploratory operation without having deglutition re-established, has not been afforded the best medical care.

RESUMEN

Se recalca a importancia de una selección del plan terapéutico en un caso dado de carcinoma del esófago. Debe haber por parte del cirujano y del médico una buena vountad y disposición para modificar un plan cuando existen desfavorables situaciones o estas aparecen. Habrá contínuamente muchos casos en los que las resecciones mayores curativas o paliativas, no puedan hacerse ya sea por mal estado general, o por imposibilidad quirúrgica.

En estos casos deben usarse otros procedimientos de menos magnitud que la resección deben llevarse a cabo.

Se obtendrá una más larga sobrevida y comodidad para mayor número de enfermos como resultado de la selección adecuada de as técnicas curativas y paliativas ahora asequibles para el tratamiento del carcinoma del esófago. El enfermo que es dado de alta con una gastrostomía ya sea porque haya rehusado la terapia del esófago o bien porque se haya hecho una exploración sin restablecimiento de la deglución, no ha obtenido del mejor cuidado médico.

RESUME

L'auteur insiste sur l'importance d'une mise au point prudente du plan thérapeutique dans un cas déterminé de cancer oesophagien. Il doit exister de la part du médecin et du chirurgien la bonne volonté et la possibilité de modifier le plan déterminé dès que 'on constate ou qu'il se produit des faits qui sembent défavorables. On doit compter encore avec un nombre important de cas pour lesquels la résection majeure, soit curative, soit palliative, ne peut avoir lieu . Elle sera empêchée ou bien par l'état gén-

éral du sujet, ou à cause de l'impossibilité de sectionner la tumeur oesophagienne. Dans ce cas, des procédés qui n'ont pas l'importance de la résection peuvent et doivent être utilisés.

Un choix meilleur des possibilités techniques actuelles pour le traitement curatif ou palliatif du cancer de l'oesophage peut permettre une plus longue survie et des conditions plus faciles à supporter pour un assez grand nombre de malades. Le malade qui quitte l'hôpital avec une gastrostomie soit que le traitement oesophagien n'ait pu être réalisé, ou bien que l'intervention exploratrice n'ait pas permis le rétablissement de la déglutition, n'a pas bénéficié du maximum des possibilités médicales.

BIBLIOGRAPHY

Adams, H. D., Hare, H. F., Davis, W. L., Trump, J. G. and Granke, R. C.: "The treatment of carcinoma of the esophagus and cardia by resection and postoperative supervoltage roentgen rays," *Ann. Surg.*, 138:631, 1953.
Allison, P. R.: "Esophagojejunostomy for irremovable carcinoma of the cardia," *Thorax*, 1:239, 1948.
Allison, P. R. and Borrie, J.: "The treatment of malignant obstruction of the cardia," *Brit. J. Surg.*, 37:1, 1949.
Berman, E. F.: "Experimental replacement of portions of the esophagus by a plastic tube," *Ann. Surg.*, 135:337, 1952.
Cliffton, E. E. and Blansfield, H. N.: "The treatment of carcinoma of the esophagus by radiation therapy and surgery," *Surgery*, 33:748, 1953.
Krebs, B., Nielsen, N. and Anderson, P. E.: "Rotation treatment of cancer of the esophagus," *Acta radiol.*, 32:304, 1949.
Lewis, I.: "The surgical treatment of carcinoma of the esophagus, with special reference to a new operation for growths of the middle third," *Brit. J. Surg.*, 34:18, 1949.
Ravitch, M. M., Bahnson, H. T. and Johns, T. N. P.: "Carcinoma of the esophagus," *J. Thoracic Surg.*, 24:256, 1952.
Souttar, H. S.: "Treatment of carcinoma of the esophagus," *Brit. J. Surg.*, 15:76, 1927.
Sweet, R. H.: "Results of radical surgical extirpation in the treatment of carcinoma of the esophagus and cardia," *Surg., Gynec. and Obst.*, 94:46, 1952.
Sweet, R. H.: "Treatment of carcinoma of esophagus and cardiac end of stomach by surgical extirpation surgery," *J. Thoracic Surg.*, 10:952, 1948.
Vinson, P. P.: "Diseases of the Esophagus, A Monograph in American Lectures in Chest Disease," *American Lecture Series Publication Number 7*, 1947.
Yudin, S. S.: "The surgical construction of 80 cases of artificial esophagus," *Surg., Gynec. and Obst.*, 78:561, 1944.

Fluoroscopy of the Chest

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A postero-anterior teleoroentgenogram of the chest is primarily a "scout" film, and diagnosis based solely on such an examination is often little more than guesswork. However, this film serves admirably in *detecting* most lesions, and aids in determining what additional studies need to be performed. Unless the clinical or roentgen diagnosis is evident, one should then proceed with further roentgen study.

As a general rule this should begin with fluoroscopy. A complete chest fluoroscopy can be accomplished within a few minutes if the examiner applies a well-organized approach. For purposes of instructing students and residents, we have divided pulmonary fluoroscopy into five phases which we have designated as *Observation*, *Rotation*, *Breathing*, *Ingestion* and *Tilting*. Their first letters spell the word *ORBIT*, which serves as a mnemonic.

1. *Observation.* After preliminary fluoroscopy of the entire chest, including the heart, attention is directed to the lesion in question. Its size, shape, homogeneity and margination are closely studied. The presence or absence of pulsation should be noted. However, this is of little significance unless opposite sides of the lesion can be seen simultaneously, since differentiation between transmitted and intrinsic pulsation cannot otherwise be made. Even under those conditions aneurysms cannot be reliably distinguished from tumors, since aneurysms often contain clot which interferes with expansile pulsation, and tumors occasionally surround a vessel and may then appear to pulsate expansively.

2. *Rotation.* By slightly rotating the patient (10 to 20 degrees) one can quickly establish whether a density lies anteriorly or posteriorly. Storch[1] utilizes the sternum and spine as reference points. If, on rotation, the lesion moves in the same direction the spine moves, it is posterior, whereas if it moves with the sternum it is anterior in location (Fig. 1). The amount of movement of the lesion indicates how far it lies from the center of the thorax (the vertical axis on which the patient is rotated). Thus, peripheral lesions shift more with rotation than central ones.

Rotation serves to separate a lesion from adjacent structures so that a clearer view of it is obtained. Obviously, if the lesion can be separated entirely from a superimposed structure by rotation, there is little likelihood that it is connected with that structure. Rotation is also helpful in differentiating hilar node enlargement from vascular shadows, the nodes remaining rounded while vascular shadows elongate or disappear as the patient is turned.

As a lesion comes closer to the fluoroscopic screen, its image appears smaller and sharper. If a density is smaller in the postero-anterior projec-

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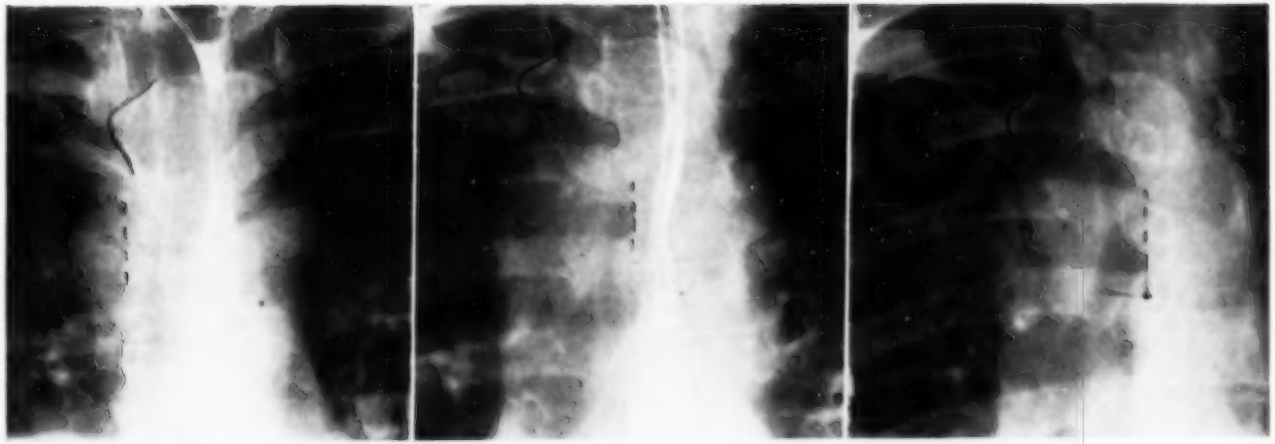

FIGURE 1A FIGURE 1B FIGURE 1C

Figure 1: Right superior mediastinal mass localized by rotation during fluoroscopy. Spot films: *A.* Straight frontal view. *B.* Ten degrees rotation to right. *C.* Twenty degrees rotation. With increasing rotation, the lateral border of the mass (retouched) shifts away from the right border of the spine (dotted line) and moves in the same direction as the manubrium (retouched), but to a slightly less degree. This indicates that the mass lies far anteriorly in the thorax. A thymic cyst was found at operation.

tion (patient facing the screen) than in the antero-posterior, it lies in the anterior half of the thorax. The patient must be in true frontal position and in contact with both table top and screen for the results of this maneuver to be valid.

Another method of localization can be utilized. With the patient facing the screen, the shutters are opened fairly wide and the screen positioned so that the image of the lesion lies on one side of the visible fluoroscopic beam. The screen is slowly shifted transversely in the direction of the lesion, which now seems to move. The greater this apparent movement, the more posterior is the lesion. Localization by this method requires considerable practice and experience, so it is not widely used. However, by taping a coin to the skin over the image of the lesion and observing the relative movement between the coin and the lesion as the screen is shifted, the interpretation is simplified.

3. *Breathing.* It is important to study the movement of the abnormal shadow as the patient breathes. During inspiration the spine remains relatively immobile while the ribs move upward, the diaphragms and lungs move downward, and the vascular markings spread apart. By careful observation during respiration it is often possible to relate a lesion to the mediastinum, thoracic wall, heart, diaphragm, or lung. A point of reference is taken in sequence on each structure in close proximity to the abnormal density, and the relative movement between this structure (rib, vertebra, segment of the heart, etc.) and the density in question is noted. If the two move together as a unit, a connection between them is postulated, but even the slightest amount of independent movement indicates that they are not intimately associated.

A recent case serves to illustrate the value of this technic (Fig. 2). A mass was discovered in the left lower posterior thorax adjacent to the

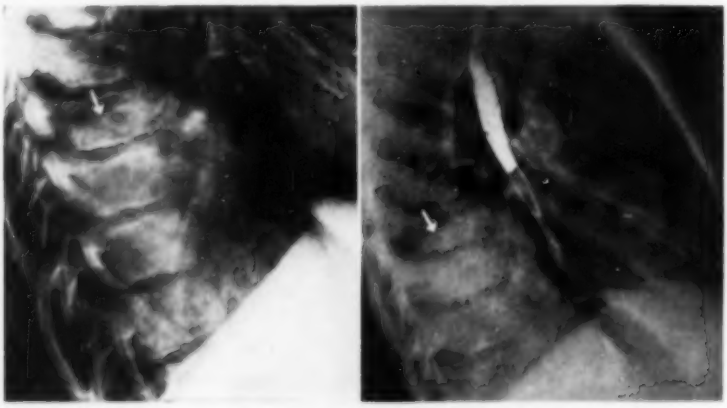

FIGURE 2A FIGURE 2B

Figure 2: Mass in posterior segment of left lower lobe. Upper border of the mass (arrow) moves slightly in relation to the overlying ninth thoracic vertebra and posterior ribs (see text). The localization was confirmed at operation. A. Inspiration. B. Expiration.

spine. Fluoroscopically, the left diaphragm showed limitation of motion. With respiration the mass moved slightly in relation to the adjacent immobile vertebral bodies and the superimposed posterior ribs. It was, therefore, concluded that the mass did not arise in the spine or chest wall, but was primarily a lower lobe lesion. This was also supported by the observation that the mass moved as a unit with the vascular markings in the adjacent lung. At operation, the mass was found in the posterior basal segment of the left lower lobe.

The effect of respiration on the heart and mediastinum should also be carefully observed. In obstructive emphysema, as well as in collapse, the mediastinal structures shift in the direction of the lesion on inspiration and in the opposite direction on expiration. The paradox of enlargement

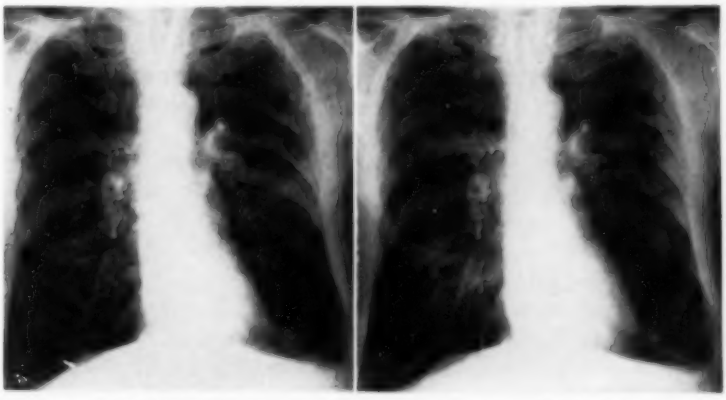

FIGURE 3A FIGURE 3B

Figure 3: Paradoxical respiratory effect on heart during an attack of bronchial asthma. Fluoroscopy revealed limited diaphragmatic excursions with minimal increase in heart size during expiration. A. Inspiration. B. Expiration.

FIGURE 4A FIGURE 4B

Figure 4A: Teleoroentgenogram in a cardiac patient with unexplained chronic pulmonary infiltrate at the right base.—*Figure 4B:* Esophago-gastric junction filled with barium, revealing an unsuspected cardiospasm. The lung changes were attributable to aspiration of esophageal contents.

of the heart shadow on inspiration and shrinkage on expiration occurs in bilateral obstructive emphysema, as seen in bronchial asthma, the "bronchiolitis" of infants, and in partial tracheal obstruction[2] (Fig. 3).

The Valsalva and Müller maneuvers,[3] coughing, sniffing, and snorting may aid in differentiating between a vascular shadow and a non-vascular mass, since alteration of the intra-alveolar pressure causes vascular structures to change in size. These technics may profitably be combined with trendelenburg and upright positions in the study of mediastinal masses.[4] They are of particular help in differentiating mediastinal lymph node enlargement from vascular shadows. Diaphragmatic excursion can often be accentuated by sniffing.

4. *Ingestion.* Ingestion of barium is an integral part of chest fluoroscopy and should never be omitted. By this means an unsuspected esophageal abnormality which is the underlying cause of the pulmonary disease may be shown (Fig. 4), or gastro-intestinal segments in the thoracic cavity, directly accounting for the abnormal roentgen appearance, may be revealed.

Evidence of impingement on the esophagus often aids in the localization of a mass lesion and may be the only indication of enlarged mediastinal lymph nodes. This is especially true of the bifurcation nodes, enlargement of which often produces an extrinsic pressure defect on the esophagus at the level of the carina, usually best seen in the left oblique view (Fig. 5). This may be the only clue to a correct pre-operative diagnosis of pulmonary carcinoma, as pointed out by Fleischner.[5]

5. *Tilting.* Examination of the patient in the prone, supine, trendelenburg, lordotic, or decubitus positions is often rewarding. The decubitus examination may be made by utilizing a stretcher in front of the upright fluoroscope, but tilting the upright patient to right and left in the frontal view, and bending at the waist in the lateral view are more practical methods. The lordotic position[6] is easily assumed during upright fluoroscopy, and is accomplished through the use of sand bags under the lower

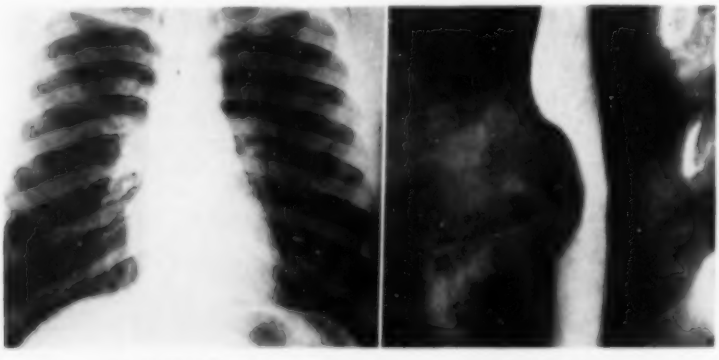

FIGURE 5A FIGURE 5B

Figure 5A: Teleoroentgenogram of chest. The right hilum is enlarged and is more dense than the left.—*Figure 5B:* Left anterior oblique view of the middle third of esophagus showing the indentation caused by enlarged carinal lymph nodes.

ribs in supine fluoroscopy. The recognition of pleural fluid, the demonstration of fluid levels, the delineation of middle lobe disease, and the displacement of pulmonary densities from overlying structures are facilitated by applying these gravitational methods.

Roentgenography

The exposure of spot films during fluoroscopy is an important part of the examination since it provides a permanent record of the findings, and brings out details that are difficult to demonstrate by other means.[7]

After fluoroscopy, additional films are obtained as indicated. The proper degree of rotation for oblique projections and the need for such positions as the lateral, lordotic, decubitus, supine, prone, and erect are determined at fluoroscopy. Expiration films are obtained when indicated by fluoroscopy, and centering for detail roentgenograms and laminagrams[8] is also facilitated.

In addition to fluoroscopy, many other roentgen methods of examination of the chest are available. Excellent descriptions of these can be found in articles by Cohen and Geffen,[9] Rigler,[10] and Robbins, Hale, and Merrill,[11] and only brief mention of them is made here. Bucky films of the chest, spine, and upper abdomen, stereoscopy, laminagraphy in frontal, lateral, and oblique planes, selective and "lung mapping" bronchography, pulmonary angiography, and diagnostic pneumothorax and pneumoperitoneum all have their specific indications.

However, there is no adequate substitute for careful chest fluoroscopy. As one gains experience with this oft-neglected method, the realization of its value in the problem case is soon appreciated.

SUMMARY

The chest teleoroentgenogram serves as a "scout" film for the detection of intrathoracic lesions. When the diagnosis is not apparent, further roentgen study of the patient is indicated, and, as a rule, this should begin with fluoroscopy.

Adequate fluoroscopic examination of a chest lesion should include the following:

1. Observation of the finer details of the lesion.
2. Rotation of the patient to determine relationships between the lesion and the adjacent normal structures.
3. Breathing maneuvers to detect vascular components and to assist in localization.
4. Ingestion of barium to ascertain origin in, or displacement of, the gastro-intestinal tract.
5. Tilting of the patient into various positions to evaluate the effects of gravity on the lesion.

Exposure of spot films during the fluoroscopic examination is essential to provide a permanent record of the findings, and additional views should

be obtained after fluoroscopy to bring out further details.

Fluoroscopy is one of the most important and rewarding methods of roentgen examination of the chest.

RESUMEN

El roentgenograma del tórax, sirve como una película de "exploración" para el descubrimiento de las lesiones intratorácicas. Cuando el diagnóstico no es aparente, se requiere un estudio ulterior y como regla éste debe empezar por la fluoroscopía.

El examen fluoroscópico adecuado debe incluir lo siguiente:

1. Observación de los más finos detalles de la lesión.
2. Rotación del enfermo para determinar las relaciones entre las lesiones y las estructuras proxímas.
3. Provocación de le respiración para descubrir la participación vascular y ayudar a la localización.
4. Ingestión de bario para aclarar el origen en o el desplazamiento del tubo digestivo.
5. Movilizar el enfermo en formas varias para estimar el efecto de la pesantez sobre las lesiones.

La-toma de películas enfocadas, durante el examen fluoroscópico es esencial para obtener un registro permanente de los hallazgos y aspectos adicionales deben obtenerse después de la fluoroscopía para mayor detalle.

La fluoroscopía es uno de los métodos más importantes y satisfactorios para el examen del tórax.

RESUME

La téléradiographie thoracique se comporte comme un élément de "reconnaissance" pour la découverte des lésions intrathoraciques. Quand le diagnostic ne s'y inscrit pas, il y a lieu de continuer les investigations radiologiques. Dans la règle, il y a lieu de commencer par la radioscopie.

Un examen radioscopique valable pour la lésion thoracique comporte les recherches suivantes:

1. Observation des plus petits détails de la lésion.
2. Rotation du malade derrière l'écran pour rechercher les relations entre la lésion et les éléments normaux voisins.
3. Manoeuvre respiratoire pour mettre en évidence une participation vasculaire et pour préciser la localisation.
4. Ingestion de baryte afin de préciser l'existence d'une lésion ou le déplacement du tractus gastro-intestinal.
5. Examen du malade en diverses positions, afin de mettre en évidence l'effet de la pesanteur sur les lésions.

La prise de clichés localisés au cours de l'examen radioscopique est essentielle pour permetttre d'avoir un témoignage permanent des constatations faites, et afin de mettre en évidence de nouveaux détails.

La radioscopie est une des méthodes les plus importantes et les plus profitables dans l'examen radiologique du thorax.

REFERENCES

1 Storch, C. B.: "Fundamentals of Clinical Fluoroscopy," New York, *Grune and Stratton*, 1951.

2 Munk, J. and Lederer, K. T.: "Inspiratory Widening of the Heart Shadow: A Fluoroscopic Sign in Acute Obstructive Laryngo-tracheitis," *Brit. J. Radiol.*, 27:294, 1954.

3 Westermark, N.: "Importance of Intra-alveolar Pressure in The Diagnosis of Pulmonary Diseases," *Radiology*, 50:610, 1948.

4 Gianturco, C.: "Intrathoracic Pressure and Position of the Patient in the Roentgen Examination of the Mediastinum," *Am. J. Roentg., Rad. Ther. and Nuc. Med.*, 71:870, 1954.

5 Fleischner, F. G.: "The Esophagus and Mediastinal Lymphadenopathy in Bronchial Carcinoma," *Radiology*, 58:48, 1952.

6 Fleischner, F. G.: "Das Röntgenbild der interlobaren Pleuritis und seine Differentialdiagnose," *Ergebn. d. med. Strahlenforsch.*, 2:197, 1926.

7 Stiehm, R. H.: "The Advantages of Roentgenoscopy and Roentgenoscopic 'Spot' Roentgenograms in the Diagnosis of Early Pulmonary Tuberculosis," *Am. J. Roentg., Rad. Ther. and Nuc. Med.*, 45:871, 1941.

8 Calder, E.: "Fluoroscopic Preselection of the Tomographic Plane in Chest Lesions with a Note on the Use of a Double Grid," *Brit. J. Radiol.*, 22:627, 1949.

9 Cohen, A. G. and Geffen, A.: "Roentgenographic Methods in Pulmonary Disease," *Am. J. Med.*, 10:375, 1951.

10 Rigler, L. G.: "The Chest. A Handbook of Roentgen Diagnosis," Chicago, *The Year Book Publishers*, 2nd ed., 1954.

11 Robbins, L. L., Hale, C. H. and Merrill, O. E.: "The Roentgen Appearance of Lobar and Segmental Collapse. I. Technic of Examination," *Radiology*, 44:471, 1945.

Combined Action of Pas and Chloromycetin on Tubercle Bacilli*

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Combined action of different anti-tuberculosis drugs has been studied by various workers. Vennesland et al (1948) studied the combined action of streptomycin and para-aminosalicylic acid and Chopra and Gupta (1952) streptomycin and chloromycetin, Gupta and Chopra (1953) chloromycetin and ptergospermin. Recently Hobby et al (1953) have reported that streptomycin and isoniazid act synergistically when used under appropriate condition in adequate quantities. The present study was undertaken to study the combined effect of PAS and chloromycetin.

PAS (Lehman 1946) is an active tuberculostatic drug which in conjunction with streptomycin delays the emergence of streptomycin resistant strains. Chloromycetin, Ehrlich et al (1947) possesses 10 times less antituberculous activity than that of streptomycin.

Chloromycetin and PAS were used.

We used the same methods as described previously (Chopra and Gupta 1952).

TABLE I

Combined action of PAS and Chloromycetin against M. tuberculosis

Conc. of PAS in ug/cc	*Concentration of Chloromycetin in-ug/cc*						
	0	10	5	2	1	0.5	0.2
0	3	0	1	3	3	3	3
10	0	0	0	0	0	0	0
8	1	0	0	0	1	1	1
5	2	0	0	1	2	2	2
2	3	0	0	1	2	2	2
1	3	0	0	1	2	2	2
5	3	0	0	1	3	3	3

Note:
0 indicates no growth
1 slight growth
2 moderate growth
3 full growth.

From table it is seen that the minimum inhibitory concentrations of PAS and chloromycetin alone are 10 ug/cc. When 8 ug/cc of PAS which inhibit the growth partially are used in combination with 5 ug/cc and 2 ug/cc of chloromycetin, there is complete inhibition of growth, while there is partial inhibition with 1 ug/cc, 0.5 ug/cc and 0.2 ug/cc of choromycetin. The results when plotted graphically (Graph 1) according to the method of Eagle et al (1948) demonstrated that the drugs act synergistically.

*From Vallabhbhai Patel Chest Institute, University of Delhi, Delhi.

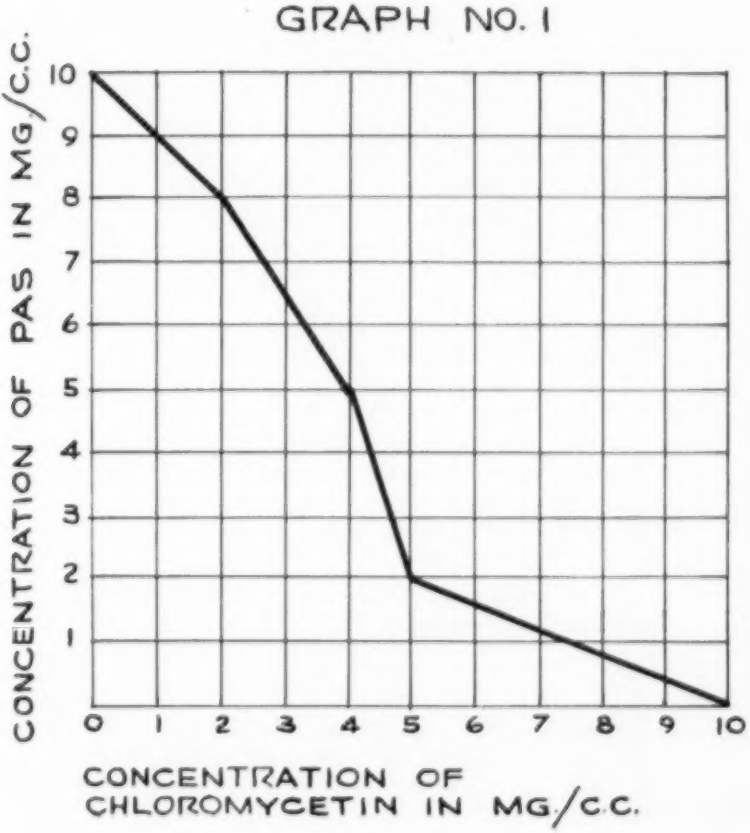
TABLE II

Combined action of PAS and Chloromycetin against bovine strain of M. tuberculosis B19-3 (Kasauli)

PAS in ug/cc	*Chloromycetin in ug/cc*								
	0	10	5	2.5	1.25	0.6	0.3	0.15	0.07
0	3	0	1	1	2	3	3	3	3
10	0	0	0	0	0	0	0	0	0
6	1	0	0	0	0	0	1	2	2
4	1	0	0	0	0	0	1	2	2
2	2	0	0	0	0	1	2	2	2
1	2	0	0	0	0	1	2	2	2
0.5	3	0	0	0	1	2	3	3	3

From the table it is seen that 10 ug/cc of PAS and 10 ug/cc of chloromycetin alone inhibit the growth, while 6 ug/cc of PAS and 5 ug/cc of chloromycetin cause partial inhibition.

Six ug/cc of PAS when used in combination with 5 ug/cc, 2.5 ug/cc, 1.25 ug/cc and 0.6 ug/cc of chloromycetin result in complete inhibition. There is partial inhibition with 0.3 ug/cc and 0.07 ug/cc of chloromycetin.



The results when expressed graphically demonstrate that the combined action of PAS and chloromycetin is synergistic (Graph 2).

SUMMARY

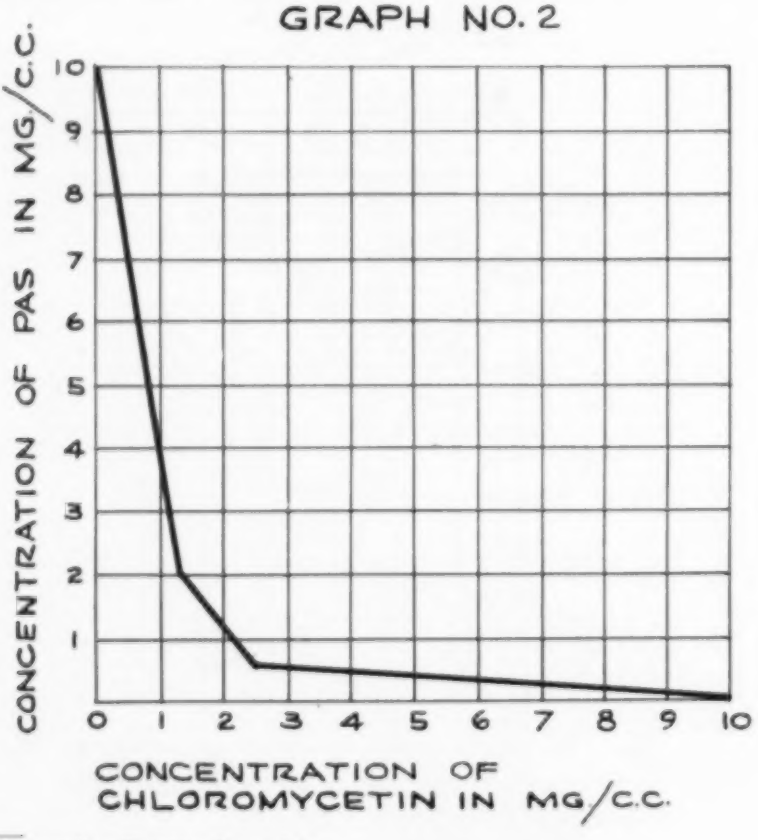
Combined action of PAS and chloromycetin has been studied against Myco. tuberculosis B19-4 (Human) and B19-3 (Bovine strains) and synergism has been demonstrated against these organisms.

RESUMEN

Se ha estudiado la acción combinada del PAS y de la cloromicetina contra la cepa de micobacteria tuberculosa B19-3 (Bovina), y se-ha demostrado sinergismo contra ambos organismos.

RESUME

Les auteurs ont étudié l'action combinée de la chloromycétine et du PAS contre la bacille de la tuberculose. Ils ont particulièrement étudié cette action contre les souches B 19-4 (souche humaine) et B 19-3 (souche bovine). Ils ont démontré que cette association avait une action synergique contre ces microbes.



Bibliography will appear in reprints.

Teaching Physical Diagnosis of the Chest

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Physical diagnosis of the chest is a dying art in this country. The information provided by skilled, available roentgenologic examination has insinuously replaced observation, palpation, percussion and auscultation as the basis for decisions in diagnosis, therapy and prognosis of many chest diseases. Perhaps this is truest for those of us who have been trained during the recent, exciting advances in laboratory methods. And yet, in our haste to employ a definitive procedure, we are vulnerable through neglect of this fundamental discipline—competent, painstaking, time consuming physical diagnosis.

All of us have studied cases in which physical diagnosis was the only accurate or available means of determining the cause of illness. Pneumonia, atelectasis, pulmonary edema, vascular anomalies and bronchial obstruction syndromes may be found by physical examination when chest x-ray films are not helpful. House officers frequently discard a diagnosis based on their physical diagnosis because roentgenograms fail to confirm their findings. Either their methods are faulty or they lack confidence in them. It can be a revealing experience to ask a student or house officer to demonstrate his concept of a complete physical examination of the chest, explaining the physiology and significance of his findings. Many of us might fare poorly also under such scrutiny. The need then, is for sounder instruction and more diligent practice of the art.

Two remedies for our fading physical diagnostic acuity are (1) performance of comfortable, careful, complete examination in all chest cases as an example to junior members of the medical echelon and (2) reorientation of our methods in teaching physical diagnosis.

It is axiomatic that medical students learn most by observing their house staff and attending physicians. Despite the number of formal presentations, the third and fourth year students are more impressed by watching internes, residents and attendings. The interne also learns from his resident and from the exceptional physician who demonstrates to him findings that he has missed and the method whereby the findings are elicited. The entire echelon profits by contact with the clinician who continually demonstrates his proficiency with physical diagnosis. But the art suffers by lack of example and by the attitude propagated at ward rounds and chest conferences where examination of the patient is replaced by perusal of x-ray films. We cannot preach the theory of physical diagnosis and practice diagnosis by x-ray films without profoundly affecting our students. It might be better to replace formal lectures by more demonstrative lessons of everyday practice.

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The third in a new series of articles prepared under the sponsorship of the Council on Undergraduate Medical Education of the American College of Chest Physicians.

A second, and chronologically earlier phase of teaching physical diagnosis is in the introductory course usually given during the second year of medical school. In some institutions this course is evolving into an introduction to internal medicine. This tendency is a natural consequence since, to understand physical findings, a knowledge of underlying dynamics is necessary. But, too often, with this approach, the patient supposedly representing a demonstration of physical findings, becomes an accessory to a discussion of disease problems. Young preceptors, whose finesse in physical diagnosis is often less than their medical acumen are most subject to this failing. A thoughtful reappraisal of each course is necessary to reorient methods to the purpose of the course.

Small groups, in which the major proportion of time is devoted to individual instruction with opportunities for practice on details of method, are effective. This approach starts with the normal subjects and utilizes repetition as the basic means to developing technique.

In the practice of a musical art or the development of athletic prowess, successful teachers devote what may seem disproportionate time to finger exercises, practice of the "correct" stroke, tackling the dummy, practicing footwork, exercises of balance and coordination. In an era when medical education has improved through application of the "learn by doing" concept, we can profit by the example of other areas where learning by doing demands rather tedious concentration on specific details.

Each student can learn best by thoroughly and repeatedly examining a classmate in a small, informal group. There is no substitute for the beginner doing it himself under the close scrutiny and criticism of his peers and their instructor. Most courses already include an hour or two of "normal" examination. However, if done correctly, this becomes a very time-consuming process, requiring more than one or two sessions in the course. The results so obtained prove that such individual instruction is necessary. To anticipate that later practice will compensate for deficiencies in technique during the learning period is the very fallacy which has produced our present problem.

The patient with abnormal physical findings must remain an important figure but is utilized for the application of techniques learned in the practice session. The students are thereby provided an opportunity to use methods they have been practicing and are introduced to abnormal findings on a background of experience with the normal. The student is encouraged to practice between classes to perfect his technique.

History taking is equally important in physical diagnosis and should receive the same individual instruction and attention.

Several ancillary lessons are taught by this approach. The students learn by experience the "feel" of a complete physical examination. Nothing else can impress upon them the subjective significance of such details as probing questions, uncomfortable positions, the nudity and inconvenience of examining gowns, clumsy technique, cold stethoscopes, cold hands and patient fatigue. Later finesse as a physician involves an appreciation of these reactions.

The President's Page

The original purpose of the American College of Chest Physicians "to bring information concerning diseases of the chest to the physician" has been the prime objective of the Committee on Scientific Program for the 21st Annual Meeting of the College to be held June 2 through 5, with headquarters at the Ambassador Hotel, Atlantic City.

During a recent visit to the College headquarters in Chicago, I had the opportunity to review the program, which appears in this issue of DISEASES OF THE CHEST. I am sure you will agree with me that it is one of the finest scientific programs of the College. The specialist in diseases of the chest and the research workers should find much to hold their attention throughout the meeting.

Some of the highlights of the program are the Selman Waksman Lecture, sponsored by the New Jersey Chapter of the College, the Fireside Conferences, panel discussions and round table luncheons. Motion picture sessions on diseases of the chest will be presented on each day of the meeting simultaneously with the scientific program. All of these special features combine to make your Atlantic City visit well worth while.

Dr. Irving Willner, Newark, New Jersey, Regent of the College, is serving as chairman of the Committee on General Arrangements for the meeting. Dr. Willner has announced that plans are progressing satisfactorily for an outstanding reunion in Atlantic City.

Thursday, June 2, will be devoted to administrative sessions and Fellowship Examinations. Councils and committees of the College will hold their annual meetings on that day. The Board of Regents and Board of Governors will meet jointly on Thursday morning, and the annual meeting of the Board of Regents will be held in the afternoon. These meetings are of prime importance to the College and its membership, as it is through the earnest and diligent work of its boards, its councils and its committees that the American College of Chest Physicians maintains its high standards and reputation for research and education in the field of diseases of the chest. I trust that every member of the College who is serving as a member of one of the councils or committees will make it a point to be present for the meetings and take an active part in the discussions. Members who are not serving on one of the councils or committees, but who have particular interest in the activities of any group, are cordially invited to join in the discussions.

The annual administrative session at which the reports of the Treasurer, Historian, Executive Director and Committee on Nominations will be received, is to be held at 9 a.m. on Saturday morning, June 4. Elections of officers of the College for the year 1955-1956 will take place at the administrative session.

On Saturday evening, June 4, the Annual Convocation of the College will be held, starting at 6:00 p.m. The cocktail party and Presidents' Banquet will follow and the evening will conclude with dancing and entertainment. Following the pattern set at our last annual meeting in San Francisco, there will be no speeches at the Annual Presidents' Banquet.

The Ladies Reception Committee, under the chairmanship of Mrs. Irving Willner, has prepared an attractive program. Will you please bring this program to the attention of your lady. We trust that she will accompany you to Atlantic City and participate in the ladies activities. Our ladies have been an inspiration to us and have added much to the success of our meetings. Mrs. Hudson and I are looking forward to the pleasure of seeing you and your lady at our 21st Annual Meeting.

William A. Hudson

21st ANNUAL MEETING PROGRAM

On the following pages we are pleased to present the scientific program for the 21st Annual Meeting of the American College of Chest Physicians, to be held at the Ambassador Hotel, Atlantic City, New Jersey, June 2-5, 1955. In organizing this program your committee made every effort to incorporate all aspects of diseases of the chest and still leave more than the usual amount of time for discussion.

In looking over the program, you will find many innovations which we trust will interest you. We wish to bring to your attention the Fireside Conferences, which are to be presented for the first time at this annual meeting. It has been the aim of the committee in organizing these Fireside Conferences, to make possble the discussion of a variety of timely topics. Our committee trusts that every member of the College will attend and participate in these conferences. They have been arranged so that everyone who wishes may take an active part in the discussion. A group of hosts will be on hand to direct the participants to the various tables. Informality and good fellowship will be the keynote of the evening's program.

The popular round table luncheons will be presented again this year. Many new subjects will be discussed by prominent scientists and, here again, there will be adequate time permitted for audience participation.

We call your special attention to the various symposia which have been organized for the meeting. The moderators of the panels have been alerted to make certain that these subjects are thoroughly discussed by the panelists and the audience.

The committee suggests that you complete the coupon for the luncheons you wish to attend and for the Presidents' Banquet. The demand for places at these functions is always far in excess of the number that can be accommodated. Our committee therefore urges that you please send in your reservations as soon as possible. You will also find a coupon for hotel reservations on page XXV.

Physicians who wish to enroll in the Seminars to be presented on Wednesday, June 1, should make application at an early date as facilities necessitate the acceptance of only a limited number. Preference, however, will be given to College members for these functions.

Our committee regrets that not all physicians who desired to participate in the 21st Annual Meeting of the College were able to be given places on the program. However, we do appreciate their interest in writing to us concerning their work.

The Committee wishes to express its thanks to the physicians who were kind enough to assist in the preparation of the scientific program and to the staff at the Executive Offices in Chicago whose assistance and cooperation have been invaluable.

Committee on Scientific Program

Burgess L. Gordon, Philadelphia, Pennsylvania, *Chairman*

Albert H. Andrews, Jr., Chicago, Illinois, *Vice-Chairman*

Alvan L. Barach, New York, N. Y.

John F. Briggs, St. Paul, Minnesota

Sumner S. Cohen, Oak Terrace, Minnesota

Alfred Goldman, Beverly Hills, California

SEMINARS

Ambassador Hotel, Atlantic City

The following seminars on diseases of the chest will be presented at the Ambassador Hotel on Wednesday, June 1, and are open to all physicians. The seminars have been accredited by the Board of Examiners for candidates for Fellowship in the College and preference will be given to members.
The registration fee for each seminar, which consists of a series of four lectures, is $7.50. Registration must be made in advance and accompanied by the tuition fee; seating capacity is limited and reservations will be accepted in the order received. A coupon for this purpose will be found opposite page 355. Please indicate your preference by number.

Wednesday, June 1

Morning Sessions—9:00 a.m.—12:00 Noon

AM-1 Diagnosis of Non-Tuberculous Pulmonary Conditions

Moderator: **Charles Hyman,** Medical Director, Atlantic County Hospital for Tuberculous Diseases, Chief of Medical Service, Atlantic City Hospital, Atlantic City

9:00 a.m. "Advances in X-ray Diagnosis"
Paul S. Friedman, Instructor in Radiology, University of Pennsylvania Graduate School of Medicine, Philadelphia

9:30 a.m. Question and answer period

9:45 a.m. "Bronchoscopic Approach in Diagnosis and Treatment"
Charles M. Norris, Clinical Professor of Laryngology and Broncho-Esophagology, Department of Laryngology and Broncho-Esophagology, Chevalier Jackson Bronchoscopic Clinic, Temple University School of Medicine and Hospital, Philadelphia

10:15 a.m. Question and answer period

10:30 a.m. "Current Status of Hypersensitivity Diseases (Collegen Vascular Diseases)"
Robert L. Mayock, Assistant Professor of Clinical Medicine (Diseases of the Chest), University of Pennsylvania School of Medicine, Philadelphia

11:00 a.m. Question and answer period

11:15 a.m. "Management of the Severe Asthmatic"
Harry L. Rogers, Assistant Professor of Clinical Medicine, Jefferson Medical College, Physician-in-Charge, Allergy Out-Patient Department, Jefferson Hospital, Philadelphia

11:45 a.m. Question and answer period

12:00 noon Adjourn for lunch

AM-2 The Physiologic Considerations of Pulmonary Disease

Moderator: **Thomas J. E. O'Neill,** Associate Professor of Thoracic Surgery, Hahnemann Medical College, Assistant Professor of Surgery, University of Pennsylvania Graduate School of Medicine, Philadelphia

9:00 a.m. "The Significance of Dyspnea"
Roger H. L. Wilson, Clinical Instructor in Medicine, University of California Medical Center, San Francisco

9:30 a.m. Question and answer period

9:45 a.m. "Evaluation of Pulmonary Function Techniques"
Richard L. Riley, Associate Professor of Environmental Medicine and Medicine, Johns Hopkins University School of Medicine and Hospital, Baltimore

10:15 a.m. Question and answer period

10:30 a.m. "Diagnostic Values of Cardiac Catheterization"
Richard T. Cathcart, Associate Professor of Medicine, Jefferson Medical College, Philadelphia

11:00 a.m. Question and answer period

11:15 a.m. "Advances in Inhalation Therapy"
Hylan A. Bickerman, Associate in Medicine, Columbia University College of Physicians and Surgeons, Research Fellow and Assistant Visiting, Columbia Research Service, New York City

11:45 a.m. Question and answer period

12:00 noon Adjourn for lunch

Wednesday, June 1, Seminars (continued)

Afternoon Sessions—2:00 p.m.—5:00 p.m.

PM-1 Pulmonary Tuberculosis

Moderator: **James S. Edlin,** Associate Clinical Professor of Medicine, New York Medical College, Tuberculosis Division, City Hospital, Manhattan General Hospital Tuberculosis Service, New York City

2:00 p.m. "The Selection of Anti-Microbial Drugs for Patients of Different Categories"
Edward H. Robitzek, Director of Medicine, Sea View Hospital, Staten Island Hospital, Staten Island

2:30 p.m. Question and answer period

2:45 p.m. "Present Status of Collapse Therapy; Medical and Surgical"
Robert V. Cohen, Assistant Professor of Medicine, Temple University School of Medicine, Philadelphia

3:15 p.m. Question and answer period

3:30 p.m. "Pulmonary Resection; Selection of Patients and Results"
Herbert C. Maier, Assistant Clinical Professor of Surgery, Columbia University College of Physicians and Surgeons, Director of Surgery. Lenox Hill Hospital, New York City

4:00 p.m. Question and answer period

4:15 p.m. "Comments on the Past and Present Methods of Prevention and Treatment"
Jacob J. Kirshner, Associate in Medicine, Jefferson Medical College, Philadelphia, Medical Director, Eagleville Sanatorium, Eagleville, Pennsylvania

4:45 p.m. Question and answer period

5:00 p.m. Adjourn to visit exhibits

PM-2 Cardiology

Moderator: **Joseph Keiserman,** Instructor in Medicine, Jefferson Medical College, Philadelphia

2:00 p.m. "The Use of Angiocardiography in Congenital Heart Disease"
George P. Keefer, Instructor in Radiology, University of Pennsylvania School of Medicine, Assistant Professor of Radiology, Woman's Medical College; **Eva F. Fox,** Associate in Medicine, Clinical Instructor in Radiology, Hospital of the Woman's Medical College, Philadelphia

2:30 p.m. Question and answer period

2:45 p.m. "Pathologic Physiology of Hypertension"
Louis A. Soloff, Clinical Professor of Medicine, Temple University School of Medicine, Philadelphia

3:15 p.m. Question and answer period

3:30 p.m. "Management of Advanced Coronary Artery Disease"
Samuel Bellet, Professor of Clinical Cardiology, University of Pennsylvania Graduate School of Medicine, Director, Division of Cardiology, Philadelphia General Hospital, Philadelphia

4:00 p.m. Question and answer period

4:15 p.m. "Newer Concepts in the Diagnosis and Treatment of Rheumatic Fever"
William I. Gefter, Associate Professor of Medicine, Woman's Medical College, Philadelphia

4:45 p.m. Question and answer period

5:00 p.m. Adjourn to visit exhibits

SCIENTIFIC PROGRAM

Thursday, June 2

7:55 p.m.—Scientific Session

William A. Hudson, President, American College of Chest Physicians, Detroit, Michigan, Chairman

James H. Stygall, President-Elect, American College of Chest Physicians, Indianapolis, Indiana, Co-Chairman

8:00 p.m.—"Evolution of the Chest Specialist"

Burgess L. Gordon, Chairman, Committee on Scientific Program, American College of Chest Physicians, President and William J. Mullen Professor of Medicine, Woman's Medical College, Philadelphia

8:10 p.m.—The Selman Waksman Lecture

Sponsored by the New Jersey Chapter, American College of Chest Physicians

"Modern Concepts of Tuberculosis Therapy"

Sir Geoffrey Todd, Medical Superintendent, King Edward VII Sanatorium, Civilian Consultant in Diseases of the Chest to the Royal Air Force; Hon. Consultant in Tuberculosis to the British Army; Consulting Physician to the Red Cross Hospital for Officers (Putney, London), Vice President, British Tuberculosis Association, Sussex, England

Symposium

"What is Causing the Increase of Cancer of the Lung?"

Moderator: **Edgar Mayer,** Clinical Professor of Medicine, New York University Graduate School of Medicine, New York City

8:30 p.m. Introduction

8:35 p.m. "Disclosure of Cancer in X-ray Surveys"

Katharine R. Boucot, Professor of Preventive Medicine and Clinical Professor of Medicine, Woman's Medical College, Chief, Division of Chronic Diseases of the Chest, Philadelphia General Hospital, Philadelphia

8:50 p.m. "Is Cancer Due to Cigarette Smoking?"

Alton Ochsner, The William Henderson Professor of Surgery and Chairman of the Department of Surgery, Tulane University of Louisiana School of Medicine, Senior Visiting Surgeon and Surgeon-in-Chief, Tulane Surgical Service, Charity Hospital, Director, Section on Surgery, Ochsner Clinic and Ochsner Foundation Hospital, New Orleans

9:05 p.m. "How About Air Pollution?"

John J. Phair, Professor of Preventive Medicine, University of Cincinnati College of Medicine; **Maurice L. Thomson,** Assistant Professor of Preventive Medicine, University of Cincinnati College of Medicine, Senior Lecturer (on leave), Department of Applied Physiology, London School of Hygiene and Tropical Medicine

9:20 p.m. "The Anatomic Approach in Answer to the Question"

Oscar Auerbach, Chief, Laboratory Service, Veterans Administration Hospital, East Orange, N. J., Associate Professor of Pathology, New York Medical College, New York City

9:35 p.m. Panel discussion and questions from the floor

10:10 p.m. Summarization by the moderator

10:20 p.m. Adjournment

Friday, June 3

8:55 a.m.—Scientific Session

9:00 a.m. "Changes of Mechanics in Breathing in Different Body Positions"
Ernst O. Attinger, Research Fellow in Medicine, Tufts College Medical School, Chief Resident, Department of Inhalational Therapy, Boston City Hospital; **Maurice S. Segal,** Clinical Professor of Medicine, Tufts College Medical School, Director, Department of Inhalational Therapy, Boston City Hospital, Boston

9:10 a.m. "The Cough Hazard"
Andrew L. Banyai, Associate Clinical Professor of Medicine, Marquette University School of Medicine, Milwaukee, Wisconsin; **Minas Joannides Jr.,** Senior Resident, Thoracic Surgical Service, Veterans Administration Hospital, Hines, Illinois

Symposium on Pulmonary Emphysema

Moderator: **Edwin R. Levine,** Assistant Professor of Clinical Medicine, Chicago Medical School, Chicago

9:30 a.m. Introduction

9:35 a.m. "Clinical Aspects"
James Kieran, Clinical Instructor in Medicine (Diseases of the Chest), Stanford University School of Medicine, Oakland, California

9:55 a.m. "Disturbances of Cardio-Pulmonary Function"
Hurley L. Motley, Professor of Medicine and Director, Cardio-Respiratory Laboratory, University of Southern California, Los Angeles

10:10 a.m. "The Role of the Diaphragm"
Coleman B. Rabin, Assistant Clinical Professor of Medicine, Columbia University College of Physicians and Surgeons, New York City

10:25 a.m. "Advances in Treatment"
Alvan L. Barach, Clinical Professor of Medicine, Columbia University College of Physicians and Surgeons, Associate Attending Physician, Presbyterian Hospital, New York City

10:45 a.m. Panel discussion and questions from the floor

11:35 a.m. Summarization by the moderator

11:45 a.m. Adjourn to visit exhibits

12:00 noon **ROUND TABLE LUNCHEON DISCUSSIONS** (see pages 349, 350 and 351)

2:00 p.m.—Scientific Session

"The Silent Zone of the Human Heart"
Myron Prinzmetal, Associate Professor of Medicine, University of California, Chief of Cardiology, City of Hope Hospital, Attending Physician, Cedars of Lebanon Hospital; **Alfred Goldman,** Chief of Professional Services, City of Hope Medical Center, Duarte, Chief, Thoracic and Cardiac Surgery, Cedars of Lebanon Hospital; **Rashid A. Massumi,** Research Associate, Cedars of Lebanon Hospital; **Louis Rakita,** Research Associate, Cedars of Lebanon Hospital, Los Angeles

Friday, June 3 (continued)

Symposium on Cardiac Manifestations in Chronic Pulmonary Disease

Moderator: **John F. Briggs**, Associate Professor of Clinical Medicine, University of Minnesota Medical School, Minneapolis

2:20 p.m. Introduction

2:25 p.m. "Physiopathology"
Julius H. Comroe, Jr., Professor and Chairman, Department of Physiology and Pharmacology, University of Pennsylvania Graduate School of Medicine; **Isaac Ott**, Professor of Physiology and Pharmacology, University of Pennsylvania Graduate School of Medicine, Philadelphia

2:40 p.m. "Evaluation of Cardiac Function and Disability"
Irving Mack, Clinical Assistant Professor of Medicine, Chicago Medical School, Associate Attending Physician, Chest Department, Michael Reese Hospital and Winfield Hospital, Research Associate, Department of Cardiovascular Research, Michael Reese Hospital, Chicago

2:55 p.m. "Special Physiologic Treatment"
Simon Dack, Lecturer in Medicine, Columbia University College of Physicians and Surgeons, Chief of the Cardiac Clinic, Mt. Sinai Hospital, New York City

3:10 p.m. "General Cardiac Treatment"
Arthur M. Master, Associate Clinical Professor of Medicine, Columbia University College of Physicians and Surgeons, Cardiologist, Mt. Sinai Hospital, New York City

3:25 p.m. Panel discussion and questions from the floor

4:20 p.m. Summarization by the moderator

4:30 p.m. Adjourn to visit exhibits

8:15 p.m.—Scientific Session

"Fireside Conferences"

Hosts:

Albert H. Andrews Jr., Chicago, Illinois
Carl C. Aven, Atlanta, Georgia
B. Guy Begin, Montreal, Canada
Otto L. Bettag, Chicago, Illinois
John F. Briggs, St. Paul, Minnesota
John S. Chapman, Dallas, Texas
Dean B. Cole, Richmond, Virginia
Seymour M. Farber, San Francisco, Calif.
M. Jay Flipse, Miami, Florida
Carl H. Gellenthien, Valmora, New Mexico
Alfred Goldman, St. Louis, Missouri
Edward A. Greco, Portland, Maine
Alvis E. Greer, Houston, Texas
Edward W. Hayes, Monrovia, California
Willard B. Howes, Detroit, Michigan
William A. Hudson, Detroit, Michigan
Hollis E. Johnson, Nashville, Tennessee
Harold I. Kinsey, Toronto, Canada
Donald R. McKay, Buffalo, New York
Herman J. Moersch, Rochester, Minnesota
Foster Murray, Brooklyn, New York
Antonio Navarrete, Havana, Cuba
James M. Odell, The Dalles, Oregon
William E. Ogden, Toronto, Canada
J. Winthrop Peabody, Washington, D. C.
Charles K. Petter, Waukegan, Illinois
Joseph C. Placak, Cleveland, Ohio
Jaime F. Pou, Hato Rey, Puerto Rico
Alfred A. Richman, New York, N. Y.
Frederick Slyfield, Seattle, Washington
James H. Stygall, Indianapolis, Indiana
W. C. Voorsanger, San Francisco, Calif.
David H. Waterman, Knoxville, Tennessee
Irving Willner, Newark, New Jersey

Friday, June 3, Fireside Conferences (continued)

Subjects and Discussion Leaders

"Emphysema"
Alvan L. Barach, Clinical Professor of Medicine, Columbia University College of Physicians and Surgeons, Associate Attending Physician, Presbyterian Hospital, New York City

"Coronary Artery Disease Surgery"
Charles P. Bailey, Professor and Head of the Department of Thoracic Surgery, Hahnemann Medical College and Hospital, Philadelphia

"Fungus Infection"
Andrew L. Banyai, Associate Clinical Professor of Medicine, Marquette University School of Medicine, Milwaukee, Wisconsin

"Angiocardiography"
Israel Steinberg, Assistant Clinical Professor of Medicine and Radiology, Cornell University Medical College, Assistant Radiologist, New York Hospital, Consultant, Veterans Administration Hospital, New York City;
Charles T. Dotter, Professor of Radiology and Head, Department of Radiology, University of Oregon Medical School, Hospitals and Clinics, Portland

"Chemotherapy"
Karl H. Pfuetze, Clinical Professor of Medicine, University of Illinois College of Medicine, Medical Director and Superintendent, Chicago State Tuberculosis Sanatorium, Chicago

"Ambulatory Treatment in Tuberculosis"
George G. Ornstein, Associate Professor of Medicine, New York Medical College, Professor of Medicine (Chest Diseases), New York Polyclinic Hospital Medical School, New York City

"Endocavitary Aspiration"
Vincenzo Monaldi, Director, Institute Principi di Piemonte, University of Naples;
Antonio Blasi, Institute Principi de Piemonte, Naples, Italy

"Asthma in Children"
Bret Ratner, Professor of Clinical Pediatrics and Head of Pediatric Allergy, New York Medical College, Director of Pediatrics, Sea View Hospital, New York City

"The Congenital Heart"
Daniel F. Downing, Associate Professor of Pediatrics, Hahnemann Medical College, Philadelphia

"Collapse Therapy"
Harold G. Trimble, Associate Clinical Professor of Medicine (Diseases of the Chest), Stanford University School of Medicine, Oakland

"Bronchoesophagology"
Chevalier L. Jackson, Professor and Head of the Department of Laryngology and Bronchoesophagology, Temple University School of Medicine, Philadelphia

"Esophageal Management"
Arthur S. W. Touroff, Clinical Professor of Surgery, Columbia University College of Physicians and Surgeons, New York City

"Extrapleural Pneumothorax"
Donato G. Alarcon, Professor of Clinical Medicine, National University of Mexico, Director, Sanatorium San Angel, Mexico City

"Bronchiectasis"
Edwin R. Levine, Assistant Professor of Clinical Medicine, Chicago Medical School, Chicago

"Inoperable Carcinoma of the Lung"
Edgar Mayer, Clinical Professor of Medicine, New York University Graduate School of Medicine, New York City

"Pulmonary Edema"
Aldo A. Luisada, Associate Professor of Medicine, Chicago Medical School, Director, Division of Cardiology, Chicago Medical School and Mt. Sinai Hospital, Chicago

"Immunity and Vaccination in Tuberculosis"
Jay Arthur Myers, Professor of Medicine and Public Health, University of Minnesota Medical and Graduate Schools, Chief, Tuberculosis Service, Minneapolis General Hospital, Minneapolis

Friday, June 3, Fireside Conferences (continued)

"Surgery in Lung Tumors"
Richard H. Overholt, Clinical Professor of Surgery (Thoracic), Tufts College Medical School, Director, Overholt Thoracic Clinic, Boston

"Rheumatic Heart Disease"
Arthur M. Master, Associate Clinical Professor of Medicine, Columbia University College of Physicians and Surgeons, Cardiologist, Mt. Sinai Hospital, New York

"Rehabilitation in Pulmonary Disease"
David A. Cooper, Professor of Clinical Medicine, University of Pennsylvania Graduate School of Medicine, Philadelphia

"Cardiac Function"
John J. Sampson, Clinical Professor of Medicine, University of California School of Medicine, Chief of the Department of Medicine, Mt. Zion Hospital, San Francisco

"Surgery of Heart Valves"
Dwight E. Harken, Associate Clinical Professor of Surgery, Harvard University Medical School, Surgeon, Peter Bent Brigham Hospital, Boston

"Excision in Tuberculosis"
J. Maxwell Chamberlain, Associate in Surgery, Columbia University College of Physicians and Surgeons, Attending in Charge of Thoracic Surgery, Roosevelt Hospital, New York City

"Pathology of Resected Lesions"
Henry C. Sweany, Director of Research, Pathology and Allied Sciences, Missouri State Sanatorium, Mt. Vernon;
Hawley H. Seiler, Director and Thoracic Surgeon, Tampa Tumor Clinic, Consultant, Thoracic Surgery, Veterans Administration Regional Office, Tampa

"Pressure Breathing"
Maurice S. Segal, Clinical Professor of Medicine, Tufts College Medical School, Director, Department of Inhalation Therapy, Boston City Hospital, Boston

"Asthma"
Leon Unger, Associate Professor of Medicine, Northwestern University Medical School, Attending Physician, Cook County and Wesley Memorial Hospitals, Chicago

"Pulmonary Function"
George R. Meneely, Associate Professor of Medicine, Vanderbilt University School of Medicine, Director, Research Laboratory and Radioisotope Unit, Veterans Administration Hospital, Nashville

"Aerosols"
Max S. Sadove, Professor of Anesthesiology and Head of the Division in Department of Surgery, University of Illinois College of Medicine, Head, Division of Anesthesiology, Research and Educational Hospitals, University of Illinois, Chicago

"Coin Shadows"
Alfred Goldman, Chief of Professional Services, City of Hope Medical Center, Duarte, Chief of Thoracic and Cardiac Surgery, Cedars of Lebanon Hospital, Los Angeles

"Rehabilitation in Cardiac Disease"
Nathaniel E. Reich, Clinical Assistant Professor of Medicine, State University of New York College of Medicine, Associate Cardiologist, Jewish Sanatorium and Hospital for Chronic Diseases, Brooklyn, New York

"Dust Diseases"
Louis L. Friedman, Consultant in Chest Diseases, Veterans Administration, Tuscaloosa, South Highlands Infirmary, Jefferson-Hillman Hospital, Birmingham, Ala.

"Positions and Incisions in Thoracic Surgery"
Emil A. Naclerio, Chief, Thoracic Surgical Services, Harlem and Columbus Hospitals, New York City

NOTE

These conferences are informal and offer an opportunity for free discussion of many subjects of interest to our members. Discussion Leaders will be seated at tables with proper identification. Physicians may participate in the discussion of their choice, or move on to other discussions when and if they desire. Refreshments will be served to enhance good fellowship.

Saturday, June 4

9:55 a.m.—Scientific Session, Section I

Unrelated Papers on Pulmonary Diseases with Special Reference to Techniques for Diagnosis and Treatment

Moderator: **Sumner S. Cohen,** Assistant Medical Director, Glen Lake Sanatorium, Oak Terrace, Instructor, University of Minnesota, Minneapolis

10:00 a.m. *Introduction*

10:05 a.m. "Clinical and Laboratory Observations in Enzymatic Therapy"
Seymour M. Farber, Associate Clinical Professor of Medicine, University of California School of Medicine; **David A. Wood,** Director, Cancer Research Institute; **Samuel L. Pharr,** Cancer Research Institute; **John K. Frost,** Instructor in Pathology, University of California Medical School, San Francisco

10:15 a.m. "Intermittent Positive Pressure Therapy in Pediatrics"
Roy F. Goddard, Director, Pediatrics Research Laboratory, The Lovelace Foundation, Albuquerque, New Mexico

10:25 a.m. "Modern Therapy and Rehabilitation in Tuberculosis"
Richard R. Trail, Medical Director, Papworth and Enhamalamein Village Settlements, London, England

10:35 a.m. Questions from the floor

10:50 a.m. "Tuberculosis and Pregnancy"
Hollis E. Johnson, Associate Professor of Clinical Medicine, Vanderbilt University School of Medicine, Nashville

11:00 a.m. "An Optimistic View of Surgery in Primary Cancer of the Lung"
Richard H. Overholt, Clinical Professor of Surgery (Thoracic), Tufts College Medical School; Thoracic Surgeon, New England Deaconess Hospital, Director, Overholt Thoracic Clinic; **James Bougas,** Overholt Thoracic Clinic, Boston

11:10 a.m. "The So-Called Tuberculoma: A Reappraisal"
J. Winthrop Peabody, Jr., Instructor in Thoracic Surgery, Georgetown University School of Medicine; **Edgar W. Davis,** Professor of Thoracic Surgery, Georgetown University School of Medicine; **Sol Katz,** Adjunct Clinical Professor of Medicine, Georgetown University School of Medicine and George Washington Medical School, Chief, Division of Pulmonary Diseases, Department of Public Health, Washington, D. C.

11:20 a.m. Questions from the floor

11:40 a.m. Summarization by the moderator

11:45 a.m. Adjourn to visit exhibits

9:55 a.m.—Scientific Session, Section II

Symposium on Cardiac Surgery

Moderator: **Charles P. Bailey,** Professor and Head of the Department of Thoracic Surgery, Hahnemann Medical College, Director, Bailey Thoracic Clinic, Philadelphia

10:00 a.m. "The Surgery of Patent Ductus and Coarctation of the Aorta"
David H. Waterman, Chief, Thoracic Surgery, Fort Sanders Presbyterian Hospital, Attending Thoracic Surgeon, East Tennessee Tuberculosis Hospital, Knoxville General Hospital, Knoxville; **William K. Rogers,** Attending Thoracic Surgeon, Fort Sanders Presbyterian Hospital, Knoxville General Hospital, East Tennessee Tuberculosis Hospital, Knoxville, Tennessee

10:10 a.m. "Tetralogy of Fallot and Pulmonic Stenosis"
John Storer, Director of the Department of Thoracic and Cardiovascular Surgery, Huron Road Hospital, East Cleveland

10:20 a.m. "Interatrial Septal Defects"
Robert E. Gross, William E. Ladd Professor of Child Surgery, Head of the Department, Children's Hospital, Harvard University Medical School, Boston

Saturday, June 4 (continued)

10:30 a.m. "Surgery of Mitral and Aortic Stenosis"
Julian Johnson, Professor of Surgery, University of Pennsylvania School of Medicine and Graduate School of Medicine, Philadelphia

10:40 a.m. "Open Cardiac Surgery: a) Hypothermia, b) Heart-Lung Apparatus, c) Cross-Circulation"
John H. Gibbon, Jr., Professor of Surgery and Director of Surgical Research, Department of Surgery, Jefferson Medical College, Philadelphia

10:50 a.m. Panel discussion and questions from the floor

11:35 a.m. Summarization by the moderator

11:45 a.m. Adjourn to visit exhibits

12:00 noon **ROUND TABLE LUNCHEON DISCUSSIONS** (see pages 349, 350 and 351)

1:55 p.m.—Scientific Session, Section I

Symposium

Is Tuberculosis in the Bag?

Moderator: **Karl H. Pfuetze**, Clinical Professor of Medicine, University of Illinois College of Medicine, Medical Director and Superintendent, Chicago State Tuberculosis Sanatorium, Chicago

2:00 p.m. *Introduction*

2:05 p.m. "Is the Virulence of the Tubercle Bacillus Changing?"
Emil Bogen, Clinical Associate Professor, Department of Infectious Diseases, University of California (Los Angeles), Head Pathologist, Olive View Sanatorium, Olive View, California

2:15 p.m. "Antimicrobial Therapy in Tuberculosis: Dosage; Resistance; and Termination of Treatment"
James A. Wier (Col.), Chief, Pulmonary Disease Service, Fitzsimons Army Hospital, Denver

2:25 p.m. "The Use of Cycloserine in the Treatment of Pulmonary Tuberculosis in Humans"
Israel G. Epstein, Associate Clinical Professor of Medicine, New York Medical College, Assistant Director, Visiting Physician, Tuberculosis Service, Metropolitan Hospital, New York City

2:35 p.m. "The Long-Term Results of Thoracoplasty Resection"
J. Maxwell Chamberlain, Associate in Surgery, Columbia University College of Physicians and Surgeons, Director of Surgery, Brooklyn Thoracic Hospital, New York City

2:50 p.m. Panel discussion and questions from the floor

3:15 p.m. Summarization by the moderator

3:20 p.m. Adjourn to visit exhibits

3:35 p.m. The Future of the Tuberculosis Sanatorium

Panel Discussion and Questions from the Floor

Moderator: **W. Edward Chamberlain**, Professor and Head of the Department of Radiology, Temple University School of Medicine, Philadelphia

Panel:

Robert J. Anderson, Assistant Chief, Division of Special Health Services, U. S. Public Health Service, Washington, D. C.
Benson Bloom, Staff, Tucson Medical Center and St. Mary's Hospital and Sanatorium, Tucson, Arizona
Edward W. Hayes, Associate Professor of Thoracic Diseases, College of Medical Evangelists and Medical Director, Maryknoll Sanatorium, Monrovia, California
Royal H. McCutcheon, Deputy Medical Director, Pennsylvania State Sanatorium, Hamburg, and Consultant, Diseases of the Chest, St. Luke's Hospital, Bethlehem, Pennsylvania
Arthur B. Robins, Director, Bureau of Tuberculosis, New York City Department of Health, Visiting Physician, Triboro Hospital, New York City

4:35 p.m. Adjourn to visit exhibits

Saturday, June 4 (continued)

1:55 p.m.—Scientific Session, Section II

Symposium

Cardiovascular Research with Special Reference to Treatment

Moderator: **William Likoff**, Associate Professor of Medicine, Hahnemann Medical College, Cardiologist, Bailey Thoracic Clinic, Philadelphia

2:00 p.m. *Introduction*

2:05 p.m. "Treatment of Cardiac Arrythmias with Grave Prognostic Import"
George E. Burch, Henderson Professor and Chairman of the Department of Medicine, Tulane University School of Medicine, Physician and Chief, Division of Medicine, Tulane Unit, Charity Hospital, New Orleans

2:20 p.m. *Discussor:* **H. Easton McMahon**, Assistant Clinical Professor of Medicine, New York Medical College, Lecturer (Cardiology), New York Polyclinic Post Graduate Medical School, New York City

2:25 p.m. "Comparative Effects of Various Digitalis Glucosides in Heart Disease"
Aldo A. Luisada, Associate Professor of Medicine, Director of the Division of Cardiology, Chicago Medical School; **Christ Aravanis**, Research Assistant; **Luigi Cardi**, Research Assistant, Chicago Medical School, Chicago

2:40 p.m. *Discussor:* **Arthur Grishman**, Instructor in Cardiology, Columbia University College of Physicians and Surgeons, Mt. Sinai Hospital, New York City

2:45 p.m. "The Over-Treatment of Apparent Sodium and Potassium Imbalance"
Isidore S. Edelman, Associate Professor of Medicine, University of California School of Medicine, San Francisco

3:00 p.m. *Discussor:* **Harry L. Jaffe**, Lecturer in Medicine, Columbia University College of Physicians and Surgeons, Assisting Attending Physician (Cardiology), Mt. Sinai Hospital, New York City

3:05 p.m. Summarization by the moderator

3:20 p.m. "Medical Therapy of Occlusive Arterial Disease"
Walter F. Kvale, Consultant, Department of Medicine, Mayo Clinic, Associate Professor of Medicine, Mayo Foundation Graduate School, University of Minnesota, Rochester

3:35 p.m. *Discussor:* **Hilton S. Read**, Director, Intern and Resident Training, Atlantic City Hospital, and Ventnor Diagnostic Center, Atlantic City

3:40 p.m. "The Techniques and Value of Vocational Guidance and Placement for the Cardiac Patient"
Herman K. Hellerstein, Clinical Instructor of Medicine, Western Reserve University School of Medicine, Assistant Physician, University Hospitals of Cleveland

3:55 p.m. *Discussor:* **William I. Gefter**, Associate Professor of Medicine, Woman's Medical College, Philadelphia

4:00 p.m. "Rehabilitation of the Cardiac Patient with Moderate Physical Limitations"
Abraham Jezer, Associate Professor of Clinical Medicine, Albert Einstein College of Medicine, Attending Physician, Montefiore Hospital; Staff, Altro Workshops, New York City

4:15 p.m. *Discussor:* **Joseph Keiserman**, Instructor in Medicine, Jefferson Medical College, Philadelphia

4:20 p.m. Summarization by the moderator

4:30 p.m. Adjourn to visit exhibits

Sunday, June 5

8:55 a.m.—Scientific Session

Symposium

Occupational Influences in Diseases of the Chest

Moderator: **Edward C. Holmblad,** Managing Director, Industrial Medical Association, Senior Attending Surgeon, St. Luke's Hospital, Chicago

9:00 a.m. *Introduction*

9:05 a.m. "Characteristic Pathology of the Lungs"
Gerritt W. H. Schepers, Director, The Saranac Laboratory, Saranac Lake, New York

9:20 a.m. "The Heart"
Richard S. Gubner, Associate Director of Medical Research, The Equitable Life Assurance Company, New York City

9:35 a.m. "Estimation of Disability and Employability"
Oscar A. Sander, Associate in Medicine, Marquette University Medical School, Milwaukee County Hospital and Columbia Hospital, Milwaukee

9:50 a.m. Panel discussion and questions from the floor

10:15 a.m. Summarization by the moderator

10:20 a.m. Adjourn to visit exhibits

10:30 a.m. **Lecture Series on Chest Diseases and Related Problems**

Moderator: **John J. Sampson,** Clinical Professor of Medicine, University of California, Chief of the Department of Medicine, Mt. Zion Hospital, San Francisco

10:30 a.m. *Introduction*

10:35 a.m. "Stimulating Opportunities for Research in Diseases of the Chest"
Herman J. Moersch, Professor of Medicine, Mayo Foundation Graduate School, University of Minnesota, Chairman, Section of Internal Medicine, Mayo Clinic, Rochester

10:45 a.m. "The Responsibility of the General Thoracic Surgeon in Cardiac Surgery"
David J. Dugan, Clinical Instructor in Surgery, Stanford University School of Medicine, Oakland, California

10:55 a.m. "Pheochromocytoma, Factors in the Accurate Pharmacologic Diagnosis"
Grace M. Roth, Consultant, Department of Physiology, Associate Professor of Experimental Medicine (Mayo Foundation), Mayo Clinic; **Walter F. Kvale,** Consultant, Department of Medicine, Mayo Clinic, Associate Professor of Medicine, Mayo Foundation Graduate School, University of Minnesota, Rochester

11:10 a.m. "The Management of Intractable Angina Pectoris"
George R. Herrmann, Professor of Internal Medicine, Director, Cardiovascular Research Laboratory, Director, Heart Station, University of Texas Medical Branch, Galveston

11:25 a.m. "How Effective are the Antibiotics against Tuberculosis?"
Edward Dunner, Chief, Training and Standards, Tuberculosis Service, U. S. Veterans Administration, Washington, D. C.

11:40 a.m. Summarization by the moderator

11:45 a.m. Adjourn to visit exhibits

12:00 noon **ROUND TABLE LUNCHEON DISCUSSIONS** (see pages 349, 350 and 351)

1:55 p.m.—Scientific Session

Lecture Series on Chest Diseases and Related Problems (continued)

Moderator: **David B. Radner,** Assistant Professor of Medicine, Chicago Medical School, Director, Chest Department, Michael Reese Hospital, Chicago, Medical Director, Winfield Sanatorium, Winfield, Illinois

2:00 p.m. *Introduction*

2:05 p.m. "Severe Crushing Injuries of the Chest: A New Method of Treatment with Continuous Hyperventilation by Means of Intermittent Positive Endotracheal Insufflation"
Edward E. Avery, Associate in Surgery, Northwestern University Medical School; **Ernst T. Morch,** Professor of Anesthesiology, University of Chicago School of Medicine; **Jerome R. Head,** Assistant Professor of Surgery, Northwestern University Medical School; **Donald W. Benson,** Instructor in Anesthesiology, University of Chicago School of Medicine, Chicago

Sunday, June 5 (continued)

2:15 p.m. "Physiological and Clinical Changes Following Closure of Atrial Septal Defects"
Harry Goldberg, Assistant Professor of Medicine and Director, Cardio-Pulmonary Laboratory, Hahnemann Medical College and Hospital; **Daniel F. Downing**, Associate Professor of Pediatrics, Hahnemann Medical College, Philadelphia

2:30 p.m. "Surgery and Supervoltage X-Ray Therapy of Cancer of the Lung"
David P. Boyd, Thoracic Surgeon, Lahey Clinic, New England Baptist and New England Deaconess Hospitals; **Carlton R. Souders**, Staff, Lahey Clinic; **Magnus I. Smedel**, Staff, Lahey Clinic, Boston

2:40 p.m. "Tuberculosis Among Nurses"
Jay Arthur Myers, Professor of Medicine and Public Health, University of Minnesota Medical and Graduate Schools, Chief, Tuberculosis Service, Minneapolis General Hospital, Minneapolis

2:55 p.m. Discussion and questions from the floor

3:15 p.m. Summarization by the moderator

3:25 p.m. Recess

3:30 p.m. Diagnostic-Treatment Conference

Moderator: **Peter A. Theodos**, Associate in Medicine, Jefferson Medical College, Chief, Diagnostic Clinic, Barton Memorial Division, Jefferson Medical College Hospital, Clinical Chief, Division of Tuberculosis, City of Philadelphia

Panel:

Internist (pulmonary)
Harold G. Trimble, Associate Clinical Professor of Medicine (Diseases of the Chest), Stanford University School of Medicine, Oakland, California

Internist (cardiology)
George R. Herrmann, Professor of Internal Medicine, Director, Cardiovascular Research Laboratory, Director, Heart Station, University of Texas Medical Branch, Galveston

Pathologist (cardiology)
Peter A. Herbut, Professor of Pathology and Head of the Department, Director of Clinical Laboratories, Jefferson Medical College and Hospital, Philadelphia

Physiologist
George R. Meneely, Associate Professor of Medicine, Vanderbilt University School of Medicine, Director, Research Laboratory and Radioisotope Unit, Veterans Administration Hospital, Nashville

Roentgenologist
Jacob H. Vastine, II, Professor of Radiology, Woman's Medical College, Associate Professor of Radiology, University of Pennsylvania Graduate School of Medicine, Philadelphia

Bronchoscopist
Albert H. Andrews, Jr., Clinical Assistant Professor of Broncho-Esophagology, Department of Otolaryngology, University of Illinois College of Medicine, Chicago

Surgeon (heart)
Alfred Goldman, Chief of Professional Services, City of Hope Medical Center, Duarte, Chief, Thoracic and Cardiac Surgery, Cedars of Lebanon Hospital, Los Angeles

Surgeon (lungs)
Wilford B. Neptune, Associate, Overholt Thoracic Clinic, Staff Member, New England Deaconess Hospital, Carney Hospital, Cambridge City Hospital, Consultant, Brooklyn Veterans Hospital, New England Hospital, Boston

The moderator will lead the discussion on two selected cases; one dealing basically with a pulmonary problem, and the other cardiac. The cases selected will represent problems commonly encountered in clinical practice and will afford an opportunity for lively discussion. The panel will have been given only a few pertinent facts in advance and each case will be presented as if currently under treatment. As the case unfolds, discussion will ensue as to the probable diagnosis, treatment and errors of omission or commission. Discussion from the floor will be invited.

ROUND TABLE LUNCHEONS

Friday, June 3

A-1 ***"Management of the Emphysematous Patient with Bronchial Asthma"***

Ethan Allan Brown, Associate Professor of Pediatrics, Tufts College Medical School, Physician-in-Chief, Allergy Section, Boston Dispensary, New England Medical Center, Boston;

A. H. Russakoff, Associate Professor of Clinical Medicine, Medical College of Alabama, Acting Chief of Tuberculosis, Veterans Hospital, Birmingham

Moderator: **Alvan L. Barach**, Clinical Professor of Medicine, Columbia University College of Physicians and Surgeons, Associate Attending Physician, Presbyterian Hospital, New York City

A-2 ***"Pulmonary Mycosis"***

Michael L. Furcolow, Associate Clinical Professor of Medicine, University of Kansas School of Medicine, Medical Director and Medical Officer in Charge, University of Kansas Medical Center, Kansas City, Kansas;

Alvis E. Greer, Clinical Professor of Medicine, Baylor University College of Medicine, Houston, Texas

Moderator: **David J. Reisner**, Visiting Physician, Sea View Hospital, Consulting Physician, Hospitals of the Department of Correction, New York City

A-3 ***"Management of Tuberculosis in the Elderly Patient"***

Kenneth G. Bulley, Medical Director and Superintendent, Kane County Springbrook Sanitarium, Aurora, Illinois;

Sol Katz, Chief, Division of Pulmonary Diseases, Department of Public Health, District of Columbia General Hospital, Adjunct Clinical Professor of Medicine, Georgetown University School of Medicine, and George Washington Medical School, Washington, D. C.

Moderator: **Harold G. Trimble**, Associate Clinical Professor of Medicine (Diseases of the Chest), Stanford University School of Medicine, Oakland, California

A-4 ***"Pulmonary Complications in General Surgery"***

Henry L. Dorfmann, Senior Clinical Chest Physician, Mt. Sinai Hospital, Attending Physician, Gouverneur Hospital, New York City;

Max S. Sadove, Professor of Anesthesiology and Head of the Division in the Department of Surgery, University of Illinois College of Medicine, Head, Division of Anesthesiology, Research and Educational Hospitals, University of Illinois, Senior Consultant in Anesthesiology, Veterans Hospital, Hines, Illinois;

Peter A. Theodos, Associate in Medicine, Jefferson Medical College, Chief, Diagnostic Clinic, Barton Memorial Division, Jefferson Medical College Hospital, Clinical Chief, Division of Tuberculosis, City of Philadelphia

Moderator: **Edward M. Kent**, Associate Professor of Surgery, University of Pittsburgh School of Medicine, Pittsburgh

A-5 ***"Bronchogenic Carcinoma: What Should be Done with the Suspect Cases?"***

Jonathan F. Meakins, Lecturer, McGill University, Associate Physician, Royal Victoria Hospital, Montreal, Canada;

Richard H. Overholt, Clinical Professor of Surgery (Thoracic), Tufts College Medical School, Thoracic Surgeon, New England Deaconess Hospital, Director, Overholt Thoracic Clinic, Boston

Moderator: **David Ulmar**, Associate Clinical Professor of Medicine, New York University Post Graduate Medical School, New York City

A-6 ***"Pericarditis"***

George R. Herrmann, Professor of Internal Medicine, Director, Cardiovascular Research Laboratory, Director, Heart Station, University of Texas Medical Branch, Galveston;

William H. Thomas, Assistant Clinical Professor of Medicine, University of California School of Medicine, San Francisco

Moderator: **William Likoff**, Associate Professor of Medicine, Hahnemann Medical College, Cardiologist, Bailey Thoracic Clinic, Philadelphia

Saturday, June 4

S-1 ***"Mass X-Ray Surveys; A Review of their Strength and Faults"***

Edward A. Piszczek, Associate Professor and Chairman of the Department of Preventive Medicine and Public Health, Stritch School of Medicine of Loyola University, Associate Professor of Public Health, University of Illinois College of Medicine, Executive Director, Suburban Cook County Tuberculosis Sanitarium District, Chicago;

Clarence M. Sharp, Director, Bureau of Tuberculosis Control, Florida State Board of Health, Consultant in Chest Diseases, Duval Medical Center, Jacksonville

Moderator: **Robert J. Anderson**, Assistant Chief, Division of Special Health Services, U. S. Public Health Service, Washington, D. C.

S-2 ***"Pressure Breathing Therapy; Positive and Negative Techniques"***

Hurley L. Motley, Professor of Medicine and Director, Cardio-respiratory Laboratory, University of Southern California, Los Angeles;

Maurice S. Segal, Clinical Professor of Medicine, Tufts College Medical School, Director, Department of Inhalation Therapy, Boston City Hospital, Boston

Moderator: **George R. Meneely**, Associate Professor of Medicine, Vanderbilt University School of Medicine, Director, Research Laboratory and Radioisotope Unit, Veterans Administration Hospital, Nashville

S-3 ***"Radioisotopes in Pulmonary Disease"***

R. Drew Miller, Consultant in Internal Medicine and Diseases of the Chest, Mayo Clinic and Rochester Methodist Hospital, Instructor, Mayo Foundation Graduate School, University of Minnesota, Rochester;

Bernard Roswit, Chief, Radiotherapy Department, Director, Radioisotope Unit, Veterans Administration Hospital, Bronx, New York

Moderator: **Allan Hurst**, Assistant Clinical Professor of Medicine, University of Colorado Medical Center, Denver

S-4 ***"Mitral Surgery; Indications and Results"***

Robert P. Glover, Clinical Professor of Thoracic Surgery, Hahnemann Medical College, Instructor in Surgery, Temple University School of Medicine, Philadelphia;

David Scherf, Professor of Clinical Medicine, New York Medical College, New York City

Moderator: **Laurence H. Rubenstein**, Department of Surgery, Chicago Medical School, Adjunct Thoracic Surgeon, Michael Reese Hospital, Consultant Thoracic Surgeon, Illinois State Hospitals, Chicago

S-5 ***"Respiratory Acidosis: Recognition and Treatment"***

Bruce Armstrong, Chief, Pulmonary Function Laboratory, Veterans Administration Hospital, Baltimore;

Edwin R. Levine, Assistant Professor of Clinical Medicine, Chicago Medical School, Chicago

Moderator: **Alexander Libow**, Associate Attending Physician, Diseases of the Chest, Mt. Sinai Hospital, Miami Beach, Florida

S-6 ***"Non-Malignant Esophageal Disease"***

Leon H. Collins, Jr., Associate in Medicine, University of Pennsylvania School of Medicine, Visiting Physician, Philadelphia General Hospital, Philadelphia;

Kenneth A. Wood, Associate Attending Physician, Grace Hospital and Detroit Chest and General Hospital, Detroit

Moderator: **Paul H. Holinger**, Professor of Bronchoesophagology, Department of Otolaryngology, University of Illinois College of Medicine, Chicago

Sunday, June 5

C-1 ***"Smog"***

Emil Bogen, Clinical Professor of Infectious Diseases, University of California (Los Angeles), Head Pathologist, Olive View Sanatorium, Olive View, California;

Heinrich Brieger, Professor and Director, Division of Industrial Medicine, Jefferson Medical College, Philadelphia

Moderator: **George Piness**, Associate Clinical Professor of Medicine, University of Southern California, Consultant, Long Beach Veterans Administration Hospital, Barlow Sanatorium, Los Angeles

C-2 ***"Does a Positive Tuberculin Test Present an Indication for Treatment?"***

Milton I. Levine, Associate Professor of Pediatrics, Cornell University Medical College, Associate Attending Pediatrician and Director of the Children's Pulmonary Clinic, New York Hospital, New York City;

James J. Waring, Professor Emeritus of Medicine, Colorado University School of Medicine, Denver

Moderator: **Julius Novak**, Associate Professor of Medicine, Chicago Medical School, Medical Director, Chicago and Cook County Tuberculosis Institute, Chicago

C-3 ***"Cor Pulmonale"***

Arthur C. DeGraff, Samuel A. Brown Professor of Therapeutics, New York University College of Medicine, Chief, New York University Cardiac Clinic, New York City;

Stephen R. Elek, Assistant Clinical Professor of Medicine, University of Southern California, Los Angeles

Moderator: **John H. Seabury**, Associate Professor of Medicine, Louisiana State University School of Medicine, Director, Lung Station, Charity Hospital of Louisiana, New Orleans

C-4 ***"The Place of Surgery in the Treatment of Coronary Artery Disease"***

Edward E. Avery, Associate in Surgery, Northwestern University Medical School, Chicago;

Samuel A. Thompson, Associate Professor of Surgery, New York Medical College, Director, Thoracic Surgery, Metropolitan Hospital, New York City

Moderator: **John F. Briggs**, Clinical Associate Professor (Internal Medicine), University of Minnesota Medical School, Minneapolis

C-5 ***"Diagnosis and Surgical Treatment of Interatrial Septal Defects"***

Osler Abbott, Assistant Professor of Surgery, Chief, Division of Thoracic Surgery, Emory University Clinic and School of Medicine, Emory University, Georgia;

Henry Swan, Professor of Surgery and Head of the Department, University of Colorado School of Medicine, Denver

Moderator: **Benjamin M. Gasul**, Clinical Associate Professor of Pediatrics, University of Illinois School of Medicine, Director, Cardio-physiology Department, Cook County Children's Hospital, Chairman, Department of Pediatrics, Presbyterian Hospital, Chicago

C-6 ***"What Should we do with Empty Beds in the Tuberculosis Sanatoria?"***

Leonard C. Evander, Assistant Medical Director, Niagara Sanatorium, Lockport, New York;

Abel Froman, Tuberculosis Consultant, Manteno State Hospital, Chicago;

Charles F. Taylor, Superintendent and Medical Director, State Sanatorium for Tuberculosis, Norton, Kansas

Moderator: **I. D. Bobrowitz**, Associate Professor of Clinical Medicine (Chest Diseases), Albert Einstein College of Medicine, Visiting Physician (Chest Diseases), Bronx Municipal Hospital Center, New York City

ADMINISTRATIVE SESSIONS

Wednesday, June 1

8:00 a.m. Registration (there is no registration fee)
2:00 p.m. Committee on Cancer
3:00 p.m. Meeting, Executive Council

Thursday, June 2

9:00 a.m. Joint Meeting, Board of Governors and Board of Regents
9:00 a.m. Committee on Occupational Diseases of the Chest
9:00 a.m. Committee on Resident Fellowships
9:00 a.m. Committee on Scientific Program
9:00 a.m. Sections, Committee on Cardiovascular Disease
10:30 a.m. Committee on College Chapters
10:30 a.m. Board of Examiners
10:30 a.m. Council on Undergraduate Medical Education
Committee on Undergraduate Medical Education
Committee on Motion Pictures
Committee on Audiovisual Aids
Committee on College Essay Award
10:30 a.m. Council on Postgraduate Medical Education
10:30 a.m. Council on Public Health
Committee on Indian Affairs
Committee on Liaison with State and County Medical Societies
10:30 a.m. Committees of the Council on Research
Non-Surgical Therapy
Pulmonary Surgery
Bronchoesophagology
Chemotherapy and Antibiotics
Physiologic Therapy
Bronchial Asthma
10:30 a.m. Committees of the Council on Hospitals
Standards and Accreditation
Rehabilitation
Hospital Statistics
Institutions
Psychosomatic Aspects

12:00 noon **LUNCHEON, 11th ANNUAL COLLEGE CONFERENCE**
(Open to all members of the College)
William A. Hudson, Detroit, Michigan, President, presiding
Awarding of Certificates of Merit to Past-Presidents of College Chapters
"Our College Chapters"
Alvis E. Greer, Houston, Texas, Chairman, Committee on College Chapters
"Undergraduate Teaching of Diseases of the Chest"
Theodore H. Noehren, Buffalo, New York, Vice-Chairman,
Council on Undergraduate Medical Education

2:00 p.m. Annual Meeting, Board of Regents
2:00 p.m. Council on Hospitals and Committees

4:00 p.m. **OPEN FORUM, COUNCIL ON HOSPITALS**
Charles A. Brasher, Mt. Vernon, Missouri, Chairman, presiding
Medical Director, Missouri State Sanatorium
"Minimum Requirements and Adequate Pay Scales for Sanatorium Personnel"
Moderator: **Edward A. Piszczek**, Executive Director, Suburban Cook County Tuberculosis Sanitarium District, Hinsdale, Illinois
Panel: **Harold G. Curtis**, Cleveland, Ohio, Medical Director, Sunny Acres Hospital and **Charles W. Scott**, Burkeville, Virginia, Medical Director, Piedmont Sanatorium
Discussion from the floor

ADMINISTRATIVE SESSIONS

Thursday, June 2 continued

4:00 p.m. Committee on Insurance

4:00 p.m. Council on Undergraduate Medical Education
- Committee on Undergraduate Medical Education
- Committee on Motion Pictures
- Committee on Audiovisual Aids
- Committee on College Essay Award

4:00 p.m. Council on Postgraduate Medical Education

4:00 p.m. Council on Public Health
- Committee on Indian Affairs
- Committee on Liaison with State and County Medical Societies

6:00 p.m. **DINNER MEETING, COUNCIL ON RESEARCH**
Open to all members of the College

Friday, June 3

4:00 p.m. Editorial Board

5:00 p.m. Committee on College Lectureships

5:00 p.m. Committee on Nominations

Saturday, June 4

9:00 a.m. Administrative Session
- Report of the Historian
- Report of the Treasurer
- Report of the Executive Director
- Report of the Committee on Nominations
- Election of Officers

Sunday, June 5

4:00 p.m. Meeting, Board of Regents

6:30 p.m. Dinner Meeting, Joint Committee on Chest X-Ray

SOCIAL ACTIVITIES

Saturday, June 4

6:00 p.m. Annual Convocation (Formal)

7:00 p.m. Cocktail Party (sponsored by The Panray Corp., New York City)

7:30 p.m. Annual Presidents' Banquet

9:30 p.m. Dancing and Entertainment
Sponsored by the New Jersey Chapter of the College

LADIES ACTIVITIES

Thursday, June 2

4:00 p.m. Tea and Reception, The Shelbourne Hotel

Friday, June 3

12:00 noon Luncheon, The Ambassador Hotel

8:00 p.m. Theater Party

Saturday, June 4

6:00 p.m. Annual Convocation (Formal)

7:00 p.m. Cocktail Party

7:30 p.m. Annual Presidents' Banquet

9:30 p.m. Dancing and Entertainment

Sunday, June 5

12:00 noon Brunch, The Traymore Hotel

MOTION PICTURE PROGRAM

"Surgical Correction of Aortic Stenosis"
Charles P. Bailey and Houck E. Bolton, Bailey Thoracic Clinic, Philadelphia, Pennsylvania

"The Use of Shunts in Aortic Arch Disease"
J. Maxwell Chamberlain, Fort Hamilton Veterans Hospital and Roosevelt Hospital, New York City

"Mechanical Factors Governing Expiratory Obstruction to Airflow"
Howard G. Dayman, Chronic Disease Research Institute, University of Buffalo School of Medicine, Buffalo, New York

"Treatment of Respiratory Conditions in Children"
Roy F. Goddard, Pediatrics Research Laboratory, The Lovelace Foundation, Albuquerque, New Mexico

"The Bronchopulmonary Segments. Part I: Anatomy and Bronchoscopy"
Chevalier L. Jackson, John F. Huber and Charles M. Norris, Temple University School of Medicine, Philadelphia, Pennsylvania (Produced by Pfizer Laboratories, Brooklyn, New York)

"Tetralogy of Fallot"
John C. Jones, University of Southern California School of Medicine, Los Angeles, California (Produced by E. R. Squibb & Sons, New York City)

"Radical Surgery for Advanced Lung Cancer"
Adrian Lambert, Columbia University College of Physicians and Surgeons, New York City

"Direct Vision Intracardiac Curative Surgery for Congenital Heart Disease"
C. Walton Lillehei, R. L. Varco, Morley Cohen and H. E. Warden, Department of Surgery, University of Minnesota Medical School, Minneapolis

"The Genesis of the Electrocardiograph and Intramural Electrocardiography"
Myron Prinzmetal, Rexford Kennamer, Rashid A. Massumi and Alfred Goldman, City of Hope Medical Center and Cedars of Lebanon Hospital, Los Angeles, California

"Gastroesophageal Resection for Carcinoma of the Lower Esophagus"
Charles B. Puestow, Veterans Administration Hospital, Hines, Illinois

"Postoperative Aerosol Therapy"
Max S. Sadove, and Reuben C. Balagot, University of Illinois College of Medicine, Chicago (Produced by Winthrop-Stearns, Inc., New York City)

TECHNICAL EXHIBITORS—21st ANNUAL MEETING

Air-Shields, Inc., Hatboro, Pennsylvania
American Cystoscope Makers, Inc., New York, New York
V. Ray Bennett and Associates, Los Angeles, California
Burnitol Manufacturing Company, Boston, Massachusetts
Warren E. Collins, Inc., Boston, Massachusetts
Joseph K. Dennis Company, Chicago, Illinois
Duncan Oxygen Therapy Company, Duncan, Oklahoma
J. H. Emerson Company, Cambridge, Massachusetts
Ethicon Suture Laboratories, New Brunswick, New Jersey
E. Fougera and Company, Inc., New York, New York
Grune & Stratton, Inc., New York, New York
Irwin, Neisler and Company, Decatur, Illinois
Eli Lilly and Company, Indianapolis, Indiana
Linde Air Products Company, New York, New York
S. E. Massengill Company, Bristol, Tennessee
Mine Safety Appliances Company, Pittsburgh, Pennsylvania
Mist O$_2$ Gen Equipment Company, Oakland, California
National Cylinder Gas Company, Chicago, Illinois
Pacific States Laboratories, San Francisco, California
The Panray Corp., New York, New York
Chas. Pfizer and Company, Brooklyn, New York
George P. Pilling & Son Company, Philadelphia, Pennsylvania
Sharp & Dohme, Inc., Philadelphia, Pennsylvania
Smith, Kline & French Laboratories, Philadelphia, Pennsylvania

TEAR ALONG EDGE

ADVANCE REGISTRATION FORM

Urgent Notice

By completing this form and returning it at once to the Executive Offices of the College in Chicago, you will avoid having to stand in line at the Registration Desk in Atlantic City. All of your credentials, as well as luncheon, banquet and other tickets, will be prepared in advance and awaiting your arrival at the hotel. Please complete both sides of this Advance Registration Form and mail promptly. Thank you.

Please Print

Name .....

Address .....

City..... State.....

Accompanied by .....

Hotel .....

Arrival date.....

Departure date .....

I am enclosing my check in the amount of $..... for reservations as indicated on the reverse side of this form.

Remarks .....

.....

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Return this form to:

Executive Offices
American College of Chest Physicians
112 East Chestnut Street
Chicago 11, Illinois

SEE PAGE XXV FOR HOTEL RESERVATION FORM

Complete the coupon on reverse side

RESERVATION FORM

Seminars, Wednesday, June 1 **($7.50 each)**

MORNING SESSIONS 9 a.m. - 12 noon	AFTERNOON SESSIONS 2:00 - 5:00 p.m.
First choice A.M.....	First choice P.M.....
Second choice A.M.....	Second choice P.M.....

Please indicate choice by number as listed in program.

Round Table Luncheons **($3.50 each)**

	Friday, June 3	Saturday, June 4	Sunday, June 5
First choice	A-.....	B-.....	C-.....
Second choice	A-.....	B-.....	C-.....
Third choice	A-.....	B-.....	C-.....

Please indicate choice by number as listed in program.

Luncheon, 11th Annual College Conference **($3.50 each)**

Thursday, June 2

I wish to reserve.....tickets for the Annual College Conference, Thursday noon, June 2.

Presidents' Banquet, Saturday, June 4 **($6.50 each)**

I wish to reserve.....tickets for the Presidents' Banquet, Saturday, June 4. (Price includes cocktails, dinner, dancing and entertainment. All seats reserved.)

Complete the coupon on reverse side

NOTE: Please make checks payable to *American College of Chest Physicians.* Checks must accompany all requests for reservations and will be accepted in the order in which they are received. All tickets will be held for pick-up at the College Registration Desk, Ambassador Hotel, Atlantic City.

TEAR ALONG EDGE

College Chapter News

ALABAMA CHAPTER

The second annual meeting of the Alabama Chapter will be held in Montgomery, April 20, preceding the meeting of the state medical association, April 21-24. The chapter will present the following program:

"Tuberculosis and Pregnancy"
Sydney Jacobs, New Orleans,, Louisiana

Carcinoma of the Lung"
John S. LaDue, New York, New York

"Follow-up on Medically Treated Fibro-caseous Nodules"
Raymond F. Corpe, Rome,, Georgia

"Broncho-pulmonary Disease with Special Reference to Pulmonary Emphysema"
Herman J. Moersch, Rochester, Minnesota

FLORIDA CHAPTER

The Florida Chapter will hold its seventh annual meeting at the Soreno Hotel, St. Petersburg, April 3, in conjunction with the annual meeting of the state medical association, April 3-6. The following program will be presented:

9:00 a.m. Business Meeting

9:45 a.m. Scientific Program

"Carcinoma of the Esophagus"
Nelson H. Kraeft, Tallahassee

"Changing Laboratory Problems in Tuberculosis"
Albert V. Hardy, Jacksonville

"Bronchoplasty in Pulmonary Tuberculosis"
Ivan C. Schmidt, West Palm Beach

"Cardiac Contusions"
W. Dean Steward and Fred Mathers, Orlando

12:00 noon Luncheon

2:00 p.m. "Mitral and Aortic Valvuloplasty"
Maurice G. Buckles, Columbus, Ohio

"Some Effects of Isoniazid Therapy in Tuberculous Diabetes"
Henry Meyer, Tampa

"Principles of Cardiac Catheterization"
F. A. Hernandez, Miami

"X-Ray Conference"
(those in attendance are requested to bring x-rays for discussion after presenting a brief and pertinent clinical history).

OHIO CHAPTER

The Ohio Chapter will meet in Cincinnati, April 20, in conjunction with the annual meeting of the state medical association. The chapter will present the following program:

"Experimental Unilateral Pulmonary Artery Occlusion in Man, with Clinical Implications"
Bernard L. Brofman, Cleveland

"The Metabolism of Isoniazid"
J. Park Biehl, Cincinnati

"The Middle Lobe Syndrome"
Laurence K. Groves, Cleveland

"Surgical Significance of Chronic Mediastinal Lymphadenitis"
Charles V. Meckstroth

NEW JERSEY CHAPTER

The New Jersey Chapter will hold a luncheon meeting at the Ambassador Hotel, Atlantic City, April 18. Preceding the luncheon, the chapter will present the following scientific program in conjunction with the Chest Section of the New Jersey State Medical Society:

10:00 a.m. "Diaphragmatic Hernia"
Adrian M. Sabety and Arthur R. Abel, East Orange

"Indications and Contraindications for Coronary Operations and the Evaluation of Coronary Surgery"
Nicholas A. Antonius, Newark

"Surgery of Coronary Heart Disease"
Alfred R. Henderson, Asbury Park

"Extent of Thoracoplasty after Pulmonary Resection"
Philip J. Kunderman, New Brunswick

"Atypical Roentgen Configuration of Pleural Effusions"
Emanuel Klosk and Arthur Bernstein, Newark

"The Problem of Solitary Circumscribed, Dense, Pulmonary Lesions"
Samuel Cohen and Frank Bortone, Jersey City

"Acute Bronchopulmonary Suppuration: Therapy with Endoscopic Application of Oleogenous Penicillin"
A. Albert Carabelli, Trenton

"Practical Testing of Pulmonary Function in the Cardio-Respiratory Laboratories of St. Michael's Hospital, Newark"
Thomas J. Ormsby, Newark

Motion Picture: "Technique of Fluoroscopy of the Chest"
Paul K. Bornstein, Asbury Park and Irving J. Selikoff, Paterson

MISSOURI CHAPTER

The annual meeting of the Missouri Chapter will be held jointly with the Missouri Trudeau Society at the Hotel President, Kansas City, March 27, in conjunction with the meeting of the Missouri State Medical Association. The following program will be presented:

12:00 noon Luncheon

1:00 p.m. "Recent Advances in Cardiovascular Surgery"
Hector W. Benoit, Kansas City, Missouri

"Human Infection with Atypical Acid Fast Organisms"
(The Yellow Bacillus)

I. "Clinical Manifestations"
Lawrence E. Wood, Kansas City, Kansas

II. "Pathology and Bacteriology"
Ann Pollak and Victor Buhler, Kansas City, Missouri

"The Medical and Surgical Management of Pulmonary Histoplasmosis"
Medical: Michael L. Furcolow, Kansas City, Kansas
Surgical: John W. Polk, Mount Vernon, Missouri

"Report on recent Veterans Administration meetings in Atlanta, Georgia, concerning chemotherapy for tuberculosis."

Speaker to be announced.

College News Notes

SEA VIEW HOSPITAL APPOINTS NEW DIRECTOR OF MEDICINE

On January 1, 1955, George G. Ornstein, M.D., F.C.C.P., after almost 25 years as Director of Medicine of Sea View Hospital, Staten Island, New York, advances to the position of Active, Honorary Consultant to the Medical Service. By election of the Medical Board, he is succeeded by Edward H. Robitzek, M.D., F.C.C.P., of Staten Island, New York.

The breadth of Dr. Ornstein's accomplishments and interests defy brief description. His organizational skill was first demonstrated when he was called to assume control of Sea View in 1931. He assembled and trained a medical staff, induced top-notch consultants to join with him in establishing consultative facilities in all the ancillary services and set patterns in the medical care of the tuberculous which have been adopted throughout the world. In addition to these accomplishments, Dr. Ornstein has found time for independent research, teaching, lecturing, writing, and the meticulous day-to-day care of myriads of tuberculous patients.

In his new consultatory capacity, Dr. Ornstein will continue his research, relieved of the time consuming problems of administration. Dr. Ornstein will also continue with his private practice in New York City. Sea View is grateful and looks forward to the continuation of his service as teacher, counsel and researcher for many years to come.

NEW ANTIBIOTIC FOR TUBERCULOSIS

Israel Epstein, M.D., F.C.C.P., Brooklyn, New York, presented a paper on a new antibiotic (*Seromycin*)* at the 14th Veterans Administration-Army-Navy Conference on the Chemotherapy of Tuberculosis in Atlanta, Georgia, on February 9. Dr. Epstein and his co-authors, Dr. K. G. S. Nair and Dr. Linn J. Boyd, revealed that *Seromycin* had been administered to 37 advanced cases of pulmonary tuberculosis over a three to four month period with the following results:

36 patients showed clinical improvement.
X-rays showed some improvement of the infected lung areas in 28 patients.
30 patients gained in weight from 4 to 14 pounds over a 16-week period.
Fever was reduced in all patients who had been febrile.
Simple smears of sputum and gastric concentrates, previously positive for the tubercle bacillus in all subjects, tested negative in 30 patients.

Twenty-nine of these cases were "chronic, far advanced, but active pulmonary cases which had failed to respond to intensive antimicrobial therapy during a year or more." Five of these were so seriously ill that the doctors continued long-standing treatment with dihydrostreptomycin and isoniazid after starting the *Seromycin*, which was given in oral doses totaling 1.0 to 1.5 grams daily. A comparison with similar cases receiving *Seromycin* alone showed no apparent advantage for the three combined antibiotics. The other 8 patients had severe tuberculosis of recent origin. They previously had received no antimicrobial drugs. Each of these showed improvement in all five categories of valuation.

Drs. Epstein, Nair, and Boyd, all of the Department of Medicine of the New York Medical College, conducted this clinical research in the Tuberculosis Division of Metropolitan Hospital, New York City.

Seromycin has shown some toxicity, being withdrawn in four cases. On the basis of their preliminary study, Dr. Epstein and his associates conclude that *Seromycin* "exerts a powerful anti-tuberculosis effect."

Seromycin was discovered by Roger Harned and Eleanor Kropp, Commercial Solvents Corporation microbiologists.

Dr. Israel Epstein will present a paper on *Seromycin* at the 21st Annual Meeting of the American College of Chest Physicians in Atlantic City on June 4, 1955.

The Veterans Administration announced that it will conduct a pilot study of *Seromycin's* clinical effectiveness. These clinical tests will be expanded if the antibiotic appears to be as useful against tuberculosis as streptomycin, PAS and isoniazid.

**Seromycin* (Trademark, Eli Lilly and Company, Indianapolis, Indiana); generic name, Cycloserine.

CAN YOU ANSWER THESE VITAL PULMONARY FUNCTION QUESTIONS?

- Why is the ratio of residual air to total lung capacity so important?
- What three important tests comprise the ventilation factor (V. F.)?
- What is the aeration gradient, and what conditions alter it?
- Why is it important to evaluate the status of carbon dioxide?
- What percentage of residual air volume indicates a severe degree of pulmonary emphysemia?
- What is the only accurate method of determining the degree of acidosis or alkalosis?
- Which factors are most often responsible for lowering arterial blood oxygen?
- Does (IPPB) for long periods of time produce increased amounts of residual air or distention of the lungs?

***NEW*—JAN., 1955 REPRINT**

by HURLEY L. MOTLEY, Ph.D., M.D., has the answers to the above questions and many more. Its title is "Physiology of Respiration with reference to Pulmonary Disease."

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CALENDAR OF EVENTS

NATIONAL AND INTERNATIONAL MEETINGS

21st Annual Meeting
American College of Chest Physicians
Ambassador Hotel
Atlantic City, New Jersey
June 2-5, 1955

Fourth International Congress on Diseases of the Chest
Council on International Affairs
American College of Chest Physicians
Cologne, Germany
August 19-23, 1956

POSTGRADUATE COURSE

2nd New Jersey
Postgraduate Course
on Diseases of the Chest
Essex House Hotel, Newark
March 9, 13, 23, 30, 1955
(four consecutive Wednesdays)

CHAPTER MEETINGS

Missouri Chapter
Kansas City, March 27, 1955

Florida Chapter
St. Petersburg, April 3, 1955

Tennessee Chapter
Chattanooga, April 12, 1955

New Jersey Chapter
Atlantic City, April 18, 1955

Alabama Chapter
Montgomery, April 20, 1955

Ohio Chapter
Cincinnati, April 20, 1955

Texas Chapter
Forth Worth, April 24, 1955

California Chapter
San Francisco, April 30, 1955

THE MEETING OF THE YEAR!

21st ANNUAL MEETING
AMERICAN COLLEGE OF CHEST PHYSICIANS

Ambassador Hotel
Atlantic City, New Jersey
June 2 through 5, 1955

104th ANNUAL MEETING
AMERICAN MEDICAL ASSOCIATION

Atlantic City
June 6-10, 1955

It is desirable that hotel reservations be completed at once for accommodations at the Ambassador Hotel, headquarters for the College meeting. If you plan to remain at the Ambassador Hotel for the meeting of the American Medical Association a deposit is required in the amount of one day's tariff, which will apply to Wednesday night, June 8. Deposit checks should be made payable to the Ambassador Hotel.

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Superintendent

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Medical Director

SPECIAL ISSUE ON CARCINOMA OF THE LUNG
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Richard H. Overholt, Boston
Ornstein and Lercher, New York City
Erik Ask-Upmark, Sweden
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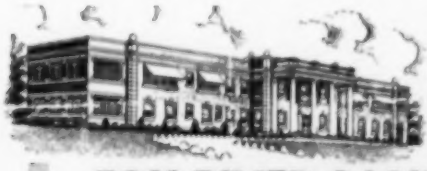
Extra copies may be obtained at the single copy price of $1.50 by writing to DISEASES OF THE CHEST, 112 East Chestnut Street, Chicago 11, Illinois, U.S.A.



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Donald F. Rowles, M.D., Oakland
James Kieran, M.D., Oakland
Robert Stone, M.D., Oakland
William B. Leftwich, M.D., Oakland

MEDICAL SERVICE BUREAU

Positions Wanted

Physician, from Spain, age 34, now residing in Central America, nine years practice in tuberculosis, experienced in general and thoracic surgery, bronchoscopy, desires position in hospital or sanatorium in United States. Good knowledge of English. Please address all inquiries to Box 291B, American College of Chest Physicians, 112 East Chestnut Street, Chicago 11, Illinois.

F.C.C.P., 34 years of age, family, desires clinic partnership or association. Available April or July, 1955. Graduate of University of Pennsylvania Medical School, 4 years training in tuberculosis, completing 3 year fellowship in internal medicine, Mayo Clinic, with special interests in pulmonary diseases. Board qualified in internal medicine. Please address all inquiries to Box 292B, American College of Chest Physicians, 112 East Chestnut Street, Chicago 11, Illinois.

Position wanted in field tuberculosis, any capacity. On staff of large sanatorium since 1938: chie , medical service, '41-'44; assistant superintendent since '44. Nearing retirement on reaching statutory age limit of 70. Native American, active, in good health, no dependents. Position and pay secondary. Address John M. Stanley, M.D., Assistant Superintendent, Glenn Dale Hospital, Glenn Dale, Maryland.

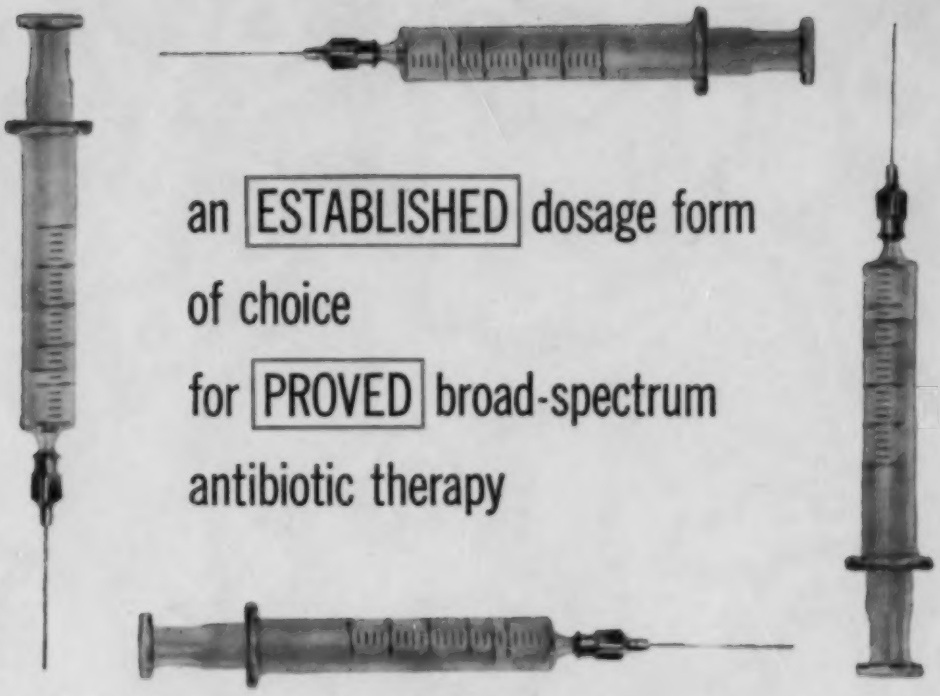
Positions Available

Applications are invited for positions of resident and assistant resident at Grace Dart Home Hospital, 6085 Sherbrooke Street, East, Montreal. This is a 147 bed tuberculosis hospital, approved by the Joint Commission on Hospital Accreditation and covers all aspects of medical and surgical treatment. Duties to commence July 1, 1955. Apply to Medical Director.

Resident physician wanted for 160 bed tuberculosis hospital in pleasant rural setting. Available July 1. All medical and surgical therapy utilized. Salary range $325 to $425 plus partial maintenance for family. Tuberculosis experience desirable but not essential. California license mandatory. Apply to Medical Director, Stony Brook Retreat, Keene, California.

Staff physician wanted for 160 bed tuberculosis hospital in pleasant rural setting. Available July 1. All medical and surgical therapy utilized. Salary range $581 to $707 plus partial maintenance for family. Tuberculosis experience desirable but not essential. California license mandatory. Apply to Medical Director, Stony Brook Retreat, Keene, California.

Medical Director wanted, North American graduate, five years tuberculosis experience, relatively new 100 bed tuberculosis hospital, Madisonville, Kentucky. Salary $10,000, complete maintenance. Apply: State Tuberculosis Hospital Commission, New State Office Building, Frankfort, Kentucky.



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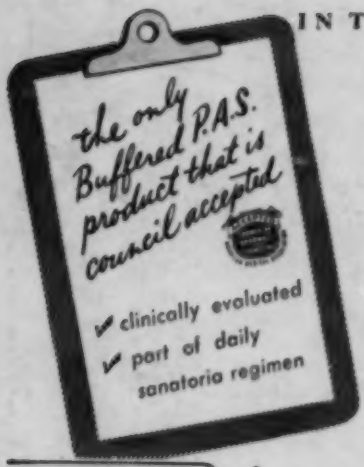

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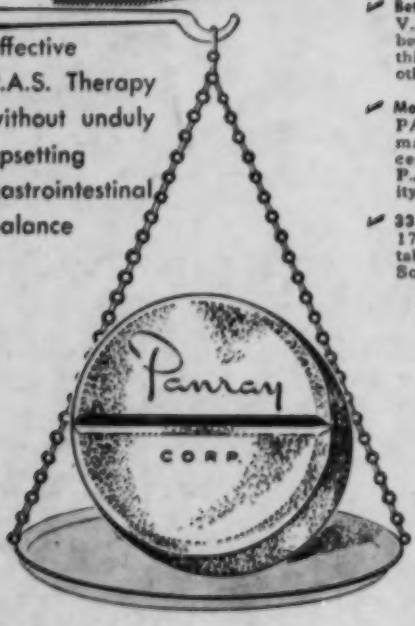

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References

(1) V.A. Quarterly Prog. Report, April '53

(2) V.A. Quarterly Prog. Report, Oct. '54

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